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CYTOLOGIC RECOGNITION OF CANCER IN EXFOLIATED MATERIAL FROM VARIOUS SOURCES

Useful Modifications of the Papanicolaou Technique

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PAPANICOLAOU'S (6) investigations of desquamated vaginal epithelium began thirty years ago with a study of the morphologic and cyclic variations of cells in the vagina of the guinea pig, and he first applied the technique to the diagnosis of uterine cancer in 1923 in the course of a study of human vaginal smears taken during the normal menstrual cycle. Despite this early recognition of the fact that cancers of the uterus could thus be diagnosed, clinical men failed to see the potentialities of the method and Papanicolaou states that he lost his enthusiasm for it until he became associated with Traut (5) with whom he jointly published the monograph *Diagnosis of Uterine Cancer by the Vaginal Smear* in 1943.

The recent war years were not conducive to widespread dissemination and application of Papanicolaou's discovery, and only a few investigators have thus far had sufficient experience to enable them to publish the results of their studies. Among these are Meigs and associates, Ayre and again Papanicolaou (6).

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Fig. 1. Vaginal smear. Only two cancer cells were found on the whole slide.

Fig. 2. Embedded material from same case as Figure 1. While only three cancer cells are present in the field others occurred elsewhere. Note that when concentrated by precipitation, many cells are seen and that all are in the same focal plane.

Fig. 3. Biopsy. Same case as Figures 1 and 2. Note how plainly the cancer cells in squamous cell carcinoma of the cervix are brought out in tissue stained by the Papanicolaou technique.

Fig. 4. Embedded and sectioned vaginal fluid. illustrating how groups of cells often remain intact and how the pattern of tissue is preserved. These are mainly basal and precornified types of squamous epithelium.

Fig. 5. Smear of bronchial mucus. A clump of cancer cells is shown but parts of it are overlaid by cells, making the interpretation more difficult.

Fig. 6. Compare the cytology with Figure 5, which is the same case. In the embedded matter there is no doubling or folding and the cytology is excellent.

Fig. 7. Bronchial aspiration, embedded. An intact segment of mucosa from a small bronchus has been obtained in an intact state.

Fig. 8. Embedded and sectioned bronchial aspiration. A portion of normal mucosa and submucosa has come off intact. Note the beautiful pseudostratification of the cells.

Fig. 9. Bronchogenic carcinoma found in bronchial mucus which was embedded and sectioned. Note the same continuity of pattern that one sees in tissue sections.

Fig. 10. Microscopic picture in mucus aspirated from the stomach. There are several normal gastric glands, separated by interglandular stroma, and one group of cancer cells. The differences between the two types is apparent.

Fig. 11. Finding in embedded mucus obtained on rectal examination. The growth was beyond the reach of the palpating finger. Histologically it proved to be an adenocarcinoma. These are cancer cells.

Fig. 12. Group of cancer cells from an instance of squamous cell carcinoma of the bladder. The cellular content of the urine was precipitated out with picric acid, filtered and embedded in paraffin.

Papanicolaou's (6) most recent communication deals not only with the smear technique as applied to uterine carcinoma, but describes its efficacy in bronchogenic, gastric, bladder, and renal cancer. It now appears that the method is applicable to cancer involving or communicating with all body orifices.

As long ago as 1935, Dudgeon and Wrigley obtained a high percentage of accurate observations of bronchogenic carcinoma, based on recognition of the cells in expectorated mucus, using Schaudinn's fluid as the fixative for the smears. Bamforth, 1946, employing the same technique as Dudgeon and Wrigley, reports satisfactory results on freshly expectorated sputum of patients with pulmonary cancer.

To date, few have attempted to modify the original Papanicolaou method of obtaining material or its preparation for study. In his last paper, concerned with identification of cancer cells in other than vaginal secretion, Papanicolaou (6) lists some modifications applicable to urine, gastric aspirations, and sputum. Ayre aspirates specimens directly from the cervix into a test tube rather than from the posterior fornix of the vagina, centrifugates, embeds in paraffin and stains all but the more questionable instances with ordinary hematoxylin-eosin.

Soon after we began to study vaginal fluid (November, 1945), both the gynecologists and ourselves became dissatisfied with some features of the Papanicolaou technique and sought to eliminate what seemed to us to be certain inherent weaknesses. In the first place, we were unable to make good even smears by blowing the content of the glass tube on slides, even though various means of smoothing out the thick areas were tried. There still remained too many dense spots and too frequent places where little or no matter was deposited. We could never convince ourselves that we were covering all fields in such smears microscopically. All too often, groups of cells were overlaid by other epithelial cells or by leucocytes; or there were thick patches through which we could not see well. Furthermore, the time required to cover a slide an inch in width and occupied by a smear covering from 2 to 2½ inches of its length was appreciable if one really attempted to see all of it.

For the first modification, most applicable to vaginal smears, we are indebted to Dr. Melvin Breese, senior resident in gynecology, whose method is as follows:

1. A warm, but dry speculum, without lubricant should be used.

2. After insertion into the vagina, enough pressure should be made on the perineum to bring the speculum to as nearly a horizontal plane as possible.

3. The fluid in the posterior fornix is gently milked into the posterior blade of the instrument.

4. The speculum is carefully removed in order to retain all possible fluid.

5. A gloved index finger is dipped into the fluid and smears are made by means of two rapid, *longitudinally* directed swipes, leaving a space at one end for labelling. It is important to avoid the natural tendency to a circular motion of the finger in placing the fluid on the slide. Such smears are invariably thick-and-thin and are quite unsatisfactory.

5. The smear is placed directly into a jar containing alcohol-ether solution as described by Papanicolaou and Traut (5).

While this was a great improvement, we were still not satisfied, for it did not eliminate the faults inherent in any smear, nor did it lessen the time required for microscopic study.

Confronted with a very thick smear made by a clinician who asked us to study his case and realizing that it would be a hopeless task, we noticed that a number of flakes had dropped off the slide into the alcohol-ether. These were recovered, embedded in paraffin, cut as one does tissue and prepared with Papanicolaou's stain. The hopelessly thick portions still adhering to the slide were shaved off with a knife and handled in the same way. To our great gratification the cytologic structure was perfect and we were able to give the doctor a diagnosis of carcinoma. It was then that we first realized the possibilities of concentrating cells, lessening the space they would occupy on a slide and the saving of time that this would bring.

Turning to possible agents which would serve the dual purpose of precipitating protein and mucus and fixation of cells, we thought of saturated aqueous solution of picric acid, one

of the best precipitants of protein known and at the same time a good fixative. Furthermore, picric acid had the advantages of easy preparation, cheapness, and noninflammability.

However, we soon learned that clinicians and nurses are not to be trusted to give the immediate care to material, so necessary if cells are to be properly preserved. For some reason too, probably because it stains, they did not take to picric acid. For this reason we still further modified the procedure by asking that those obtaining specimens of bronchial aspirations, gastric fluid, rectal mucus, ascitic or other fluid and urine, immediately add a good quantity of 10 per cent formalin to the material. Formalin is always available and there is no excuse for not using it. This step serves to arrest cellular autolysis and at least begins the process of fixation. When the material reaches the laboratory the technician then adds a quantity of saturated aqueous solution of picric acid, filters through filter paper, presses the precipitate into the tip of the cone with a rod until it becomes a compact ball and then handles it like tissue by embedding in paraffin.

The results have been most gratifying. Cells of all kinds are well preserved; they are concentrated, obviating the necessity of long search over wide areas; the Papanicolaou stain can be employed just as successfully as with cells fixed in alcohol-ether; all cells are in the same focal plane and do not overlie one another; a number of sections can be cut and mounted on a single slide, assuring an adequate sampling at different levels and affording economy in slides. We have found that for most embedded material less than five minutes is necessary to survey the section under low power and rarely more than ten minutes is required for the high power study. Finally, and most important of all, groups of cells are not torn apart as is very likely to happen with smearing, thus enabling one to see cells in pattern and in relation to their fellows.

Vaginal fluid. We generally adhere to the original Papanicolaou technique of making a smear at the time the material is obtained and of fixing it immediately in alcohol-ether. The remainder of the specimen is not discarded, however. It is carefully run into a large calibered test tube (taped to the bottle for receiving the slide) containing 70 per cent alcohol. Or, if the gynecologist wishes, he may put the remainder directly into the alcohol-ether along with the slide. In either case, care should be taken to see that the mass is kept in one piece as it slides into the fluid and that the container be handled gently so that the matter does not break up into small pieces, requiring centrifugation, which is extra work.

It is true that material obtained by milking out the posterior fornix with the speculum often contains sheets of cells which are probably scraped off with the instrument but this is not a disadvantage because the cells, whether normal or abnormal, exhibit the continuity that pathologists so much like to have. Even in smears we frequently find such sheets and in the embedded material these are still more common, since there has been no opportunity for disrupting them.

Another feature requiring mention has to do with the staining qualities of cellular cytoplasm in vaginal fluids whether fixed *en masse* or in alcohol-ether or precipitated out with picric acid. In material fixed on slides in ether-alcohol the cytoplasm of basal cells stain blue to brown but when fixed as tissue the different varieties of squamous epithelium stains red to pink. In our opinion this is not a drawback for one can readily recognize the different types of squamous epithelium by size and nuclear characteristics. When the method is confined to cancer study the staining reaction of the cytoplasm is of no importance.

It should also be mentioned that cells which have been embedded frequently lie on edge rather than flat, as in smears. Furthermore, it is common to see a clear halo or an apparent vacuole in the flat-lying cells. These differences between smears and sections are quickly appreciated and are readily evaluated.

Bronchial mucus. To obtain a good biopsy through the bronchoscope is often difficult and in instances where the tumor is inaccessible to the instrument, it is impossible. To the bronchoscopist, then, any means of obtaining additional information should be welcomed.

Thus far we have confined our observations to secretion obtained at the time of bronchos-

copy and have not worked with sputum which has been expectorated.

The means of collection of mucus is simple. The bronchoscopist saves the content of the trap, which may be submitted directly to the laboratory, or pours the material into alcohol-ether or formalin. In our experience it is difficult to make good smears of thick mucus and we have largely given it up in favor of fixation, embedding, sectioning, and staining with the Papanicolaou stain.

Apparently, cells of all kinds are well protected and do not break down quickly in bronchial secretion, a feature that does not obtain with gastric mucus, which must be fixed quickly.

It is surprising, particularly in embedded mucus, how often sizeable sheets of mucosa or neoplasm are loosened by the bronchoscope and also how frequently one finds masses of bacteria clinging to dead epithelial cells like a swarm of bees. Another surprise is the frequency with which blood pigment laden monocytes are present in bronchial secretion of persons without evidence of heart failure. The bacterial flora also stands out very distinctly. There is little that need escape attention and recognition if it happens to be incorporated in mucus aspirated from bronchi.

It is not difficult to recognize bronchial epithelium whether squamous or pseudostratified columnar, in groups or singly. Normal columnar cells, cut longitudinally, have basally placed nuclei with scattered chromatin granules and coarsely granular acidophilic cytoplasm. The more superficial cells are apt to appear quite broad and "fat" while the deeper ones are more typically columnar. Cells cut transversely, whether singly or in masses, are likely to display little cytoplasm and thus may be mistaken for carcinoma unless weight is given to the uniformity in the size of the nuclei and the amount and distribution of the nuclear chromatin. We have found it easier to distinguish normal bronchial epithelium from carcinoma than it is to differentiate between endometrium and corporal adenocarcinoma.

By the simple procedure outlined we have been able to give valuable aid to bronchoscopists on a number of occasions. Practically always when the biopsy is positive for carcinoma, the embedded mucus will also contain tumor cells and thus serves as a check. If the biopsy is very small or fails to include enough tumor to make it satisfactory for diagnosis, the help given by cells in mucus is very real.

A recent case will illustrate the point. A patient with an 8 centimeter sized shadow in the roentgenogram of one lung was subjected to bronchoscopy. Nothing was visible in any bronchi accessible to the instrument. A biopsy taken from the orifice of a lateral bronchus of the second order, proved to be normal mucosa and submucosa. The mucus aspirated from the affected side contained enough cells to enable us to state positively that bronchogenic carcinoma was present. Pneumonectomy was performed and the diagnosis was confirmed both as to the existence of carcinoma and the cell type which had been found in the mucus.

Gastric aspiration. This may be obtained by passing a stomach tube, preferably when the viscus is empty, or at the time of gastroscopy. The gastroscopist should aspirate either from visible lesions or their vicinity whenever possible. When properly obtained and intelligently handled, much valuable information can be gained from a cytologic study of gastric secretion. Heretofore the gastroscopist has had to depend upon visual observation and interpretation. Now a valuable and easily applied adjunct is at his command.

The presence of digestive ferments makes it imperative that gastric mucus be very promptly fixed, either in formalin or in picric acid solution. It is next to impossible to make thin smears of this extremely viscid mucus, and we no longer attempt to do so. Embedding and sectioning, on the other hand, are very satisfactory.

The cytology of gastric content is both surprising and interesting. It is amazing how often the bulk of cells, or even all of them, are of squamous type. Presumably these have been swallowed and come from the mouth and esophagus. As with bronchial secretion, one often finds that the stomach tube or the gastroscope detach small and intact segments of gastric mucosa.

Single, normal gastric epithelial cells are columnar, with clear, light pink staining cyto-

plasm and rather dark but small and basally placed nuclei. Cells cut transversely seem to be almost all nucleus, but uniformity in size, shape and staining reaction differentiate from carcinoma. Gastric carcinoma cells are as readily identified as are those in bronchial or uterine cancer.

Urine. Thus far we have studied only known instances of carcinoma of the bladder but have found, as have others, that the cytologic differences between normal squamous mucosa and carcinoma here is as clear cut as in other locations.

It is most important that only freshly passed urine be employed and further, to inhibit cellular disintegration, to add formalin solution to the specimen as quickly as possible. Picric acid is equally good if it is at hand.

We do not make smears of urine; instead it is precipitated with picric acid, filtered and the product embedded.

Rectal mucus. Although our experience is quite limited in so far as the study of material derived from other than operative specimens of carcinoma is concerned, we believe that because both normal and neoplastic epithelium is well preserved for a time in lower colonic mucus, this is another location to which the smear method is readily applicable. Apparently, well developed and fungating carcinomas of the colon shed many cells, often in the form of small masses, which become incorporated in mucus and are easily found in sections of embedded material (Fig. 11, frontispiece).

Specimens of mucus may be obtained from the examining finger, the anoscope or the proctoscope, or even from the surface of a formed stool.

The normal cytology is readily established and one soon distinguishes non-neoplastic cells by their columnar form, uniformity in size and staining reaction of the nuclei, just as in the stomach and bronchi.

Transudates and exudates. Much time and effort can be saved by the simple procedure of precipitating protein from fluid with picric acid, filtration, embedding, and sectioning.

The concentration of cells, so easily obtained by this method, enables one to gain a good idea of the cytology in a short space of time. We have found that the Papanicolaou stain is as much superior to hematoxylin-eosin for cells in fluids as it is for mucus or other material.

It is common knowledge that in actinomycosis, demonstration of the organism is not easy after draining sinuses have been established. Heretofore, it has been necessary either to search repeatedly for the "sulphur granules" in pus or to curette the granulation tissue from the sinuses and section it in order to demonstrate the actinomyces. In a case coming to our attention recently we simply took a quantity of the pus, allowed it to drip into picric acid and in sections of the many masses thus obtained it was not at all difficult to find the organism.

The value of the Papanicolaou stain in the recognition of all sorts of normal and abnormal cells cannot be overemphasized. In our hands it has proved to be far superior to other tissue stains, chiefly because of the striking sharpness with which the nuclei stand out as one surveys the cells under low power and the really fine nuclear detail brought out by higher magnification. Furthermore, the stain is singularly free from technical difficulties and can be done by any tissue technician.

SUMMARY

1. The cytologic recognition of carcinoma in exfoliated material, originally described by Papanicolaou, is reliable in the hands of experienced cytologists and can be applied to a number of organs and systems of the body.

2. Not only is this method of value in the diagnosis of cancer of the uterus, where it was first applied, but also to carcinoma of the bladder, rectum, stomach, and bronchus.

3. The method is a valuable adjunct to the bronchoscopist and to the gastroscopist in particular. We believe that the mucus obtained at bronchoscopy and gastroscopy should always be saved, embedded, and sectioned. In a number of instances already we have been able to make a positive diagnosis of bronchogenic carcinoma from cells found in the mucus when the biopsy was negative or equivocal.

4. A modification of the method of obtaining vaginal fluid which, in our hands at least, has proved to be easy and workable is described. Concentration of cells saves much

time in study and artifacts are practically eliminated. The Papanicolaou stain can be applied just as readily to such material as to smears.

Addendum. In our work we employ isopropyl alcohol in the ether-alcohol fixation of smears, as a solvent for certain of the stains and as a dehydrating agent. It may be presumed from his writings that Papanicolaou uses ethyl alcohol, although he does not so state.

In general it may be stated that isopropyl alcohol tends to make the cytoplasm of cells stain acidophilic which would be basophilic if ethyl alcohol were used.

When material is initially fixed in formalin, it is most important that picric acid be superimposed. Otherwise the cytoplasm of cells is prone to be deeply basophilic and does not contrast well with the nucleus, a factor of great importance when the low power microscopic survey is being made.

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TUMORS OF THE CAROTID BODY

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THE first carotid body tumor was removed by Riegner in 1880 and described by Marchand in 1891. The carotid artery, the jugular vein, the vagus, hypoglossal, and sympathetic nerves were divided. The patient died on the third postoperative day. In 1866 Madyl and Gersuny each excised a carotid body tumor, with recovery of the patient. The common, external, and internal carotid arteries were divided in each instance with resulting hemiplegia in the case reported by Madyl. Albert, in 1889, excised the first carotid body tumor without sacrifice of major vessels, but the tumor recurred within 1 year. Collective reviews by Keen (1906), Reid (1920), Royster (1924), Phelps, Case, and Snyder (1937), Bevan and McCarthy (1929), and Gratiot (1943) have served to point up the many problems relating to the diagnosis and treatment of this disease. From these reviews it is reasonable to estimate that not more than 300 authentic cases of tumor of the carotid body have been recorded.

The carotid bodies, first described by Von Haller, in 1743, are 2 in number, situated near the bifurcation of each carotid artery. Their development proceeds until the age of 20 or 30 years, followed by a period of quiescence, and finally by sclerosis and atrophy.

They vary in size within narrow limits, but usually measure about 5 millimeters in their greatest diameter. The normal gland is gray or reddish brown in color. It is attached by a fine vasculofibrous pedicle to one of the carotid arteries, usually the external, from whence it derives its nutrient vessel. The structure is encompassed by a fibrous capsule from which projections penetrate the substance of the gland, dividing it into lesser lobules.

The individual cells are polygonal, the protoplasm is finely granular, and the large oval nucleus contains a small amount of chromatin. The cells generally are arranged in alveolar fashion. Some of the cellular elements exhibit an affinity for the chromic salts and this feature has invited speculation about their functional relationship to the chromaffin system.

The pathology of the carotid body is limited to tumor formation. These tumors have been designated adenomas, peritheliomas, endotheliomas, hamartomas, paragangliomas, neuroblastomas, and more simply, carotid body tumors. With such lack of unanimity regarding nomenclature it seems preferable to employ the latter term.

Grossly, the tumors tend to retain the shape, lobulation, and encapsulation of the normal carotid body. They are firm, compressible, slow growing tumors which eventually at times encircle the carotid vessels on the affected side. They vary considerably in size, with the largest tumor, reported by Reid, weighing 190 grams.

Microscopically, according to Harrington, Clagett, and Dockerty, the tumors exhibit two cellular patterns, occurring either alone or in combination. One pattern they designate "alveolar" and the other "perithelioma-tous." The former is characterized by large islands of pale polyhedral cells, separated by a small amount of connective tissue. In the latter arrangement, the cellular groups are smaller, irregular, and separated by an abundant, vascular connective stroma. Giant and plasma cells may be found, as well as areas of degeneration and hemorrhage.

Malignant manifestations, such as variation in size and shape of individual cells, mitoses, and invasion of the capsule and adjacent structures occur in 15 to 20 per cent of cases (Gratiot). The distinction between benign and malignant tumors of the carotid body may be difficult, and it is imperative, therefore, that adequate biopsy specimens be secured for the pathologist.

Local metastases to regional nodes have been demonstrated, but distant metastases, suspected in several instances, including 1 of our cases, have not been verified.

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TABLE I.—RESULTS

	Expired	Recovery
With ligation of common, internal, and external carotids	2	4
With ligation of external carotid artery		2
Without ligation	1	4
Exploration only		4
Subtotal excision		1
Totals	3	15

Carotid body tumors, while by no means common, are by no means rare. They have two distinctly undesirable qualities. They become malignant in a percentage which, while debatable, is disturbing. They occasionally so surround the common, external, and internal carotid arteries and are so fused with them that their removal can be accomplished only by ligation of these vessels, a procedure which in our experience has associated with it a mortality and morbidity which is forbidding.

While there have been numerous reports on these tumors, no one has had large numbers of them to deal with since approximately 300 cases comprise the total number reported in a series of articles reviewed on the subject from 1891 to 1943.

Because of the facts stated, we wish to report on 18 carotid body tumors observed at this clinic, to discuss from this experience some of their diagnostic features, to make some observations upon their surgical removal and the problems involved, to report upon the percentage of malignancies in our series and upon the mortality and morbidity following their removal. As the result of this experience, we would like also to present our position regarding the preparation of these patients for surgery, particularly as relates to possible ligation of the carotid vessels and as to when we would or would not attempt removal of the tumor.

The four laterally located tumors of the neck which are single, discrete, and movable in character, are branchial cysts, carotid body tumors, single large lateral aberrant thyroid, and neurofibromas. They are found, in our experience, in about the order stated in frequency of occurrence.

TABLE II.—SYMPTOMS

Swelling	18
Gradual increase in size	12
Pain in neck and/or face	5
Hoarseness	3
Dysphagia	1

The most frequent differentiation which will have to be made is between branchial cyst and carotid body tumor, and while this differentiation will be at times difficult, if not impossible, there are some quite distinguishing features to each of these conditions.

As anyone knows who has operated upon many branchial sinuses, these tracts are superficial in location. They are so superficial that in most instances, with traction upon the fistulous opening, so often at the level of the cricoid and just in front of the sternomastoid, the branchial sinus tract can be made to stand out so that it can be readily palpated directly under the skin until it disappears under the digastric muscle to connect with the lateral pharyngeal wall in the region of the tonsil. It is for this reason that branchial cysts, forming as they do so frequently in the lower half of the tract, are so superficial in character and located as a rule well below the level of the division of the carotid and tend as they enlarge to bulge outward upon the neck and become more and more superficial (Fig. 1).

In contradistinction to this, carotid body tumors, located as they are in the carotid notch, unlike branchial cysts originate at a higher level and are more deeply located. As they enlarge, they do so upward under the angle of the jaws and at times inward until, as we have seen in 2 of our cases, they bulge into the pharynx and, as happened in 1 of the cases, result in interference with swallowing (Fig. 2).

The other single laterally located neck tumor in this region which can be confused with a carotid body tumor is a single lateral aber-

TABLE III.—FINDINGS

Firm mass below angle of jaw	18
Compressible	3
Thrill and bruit	3
Cranial nerve pathology	2
1 hypoglossal	
1 recurrent laryngeal	

TABLE IV.

Sex		Side	
Male	9	Right	8
Female	9	Left	7
		Bilateral	3

2nd tumor not verified by exploration in 2 instances

Fig. 1. Both patients have branchial cysts. Note their superficialness, their tendency to enlarge outward and the fact that their greatest diameter is below the level of the carotid notch.

rant thyroid tumor. Lateral aberrant thyroid tissue originating from the ultimobranchial bodies at the beginning of the seventh week of the embryo is set free from the pharynx and develops contact with the two lobes of the thyroid. In occasional cases the ultimobranchial bodies become converted into thyroid tissue and form either a single lateral mass or masses located in front of the sternomastoid muscle along the course of the internal jugular vein. When these are multiple, gland-like structures, as they most frequently are, there is no question of their being confused with a carotid body tumor. When, however, they occur as a single discrete, laterally located, movable tumor mass, as they occasionally do, they can and have been confused with a carotid body tumor. Such a single lateral aberrant thyroid tumor occurred in 1 of our patients in whom we removed a carotid body tumor from the other side, and was mistakenly diagnosed by one of us (F.H.L.) as a bilateral carotid body tumor until removed and sectioned. These tumors are often fairly deep in location, discrete in character, movable, and differ from carotid body tumors only

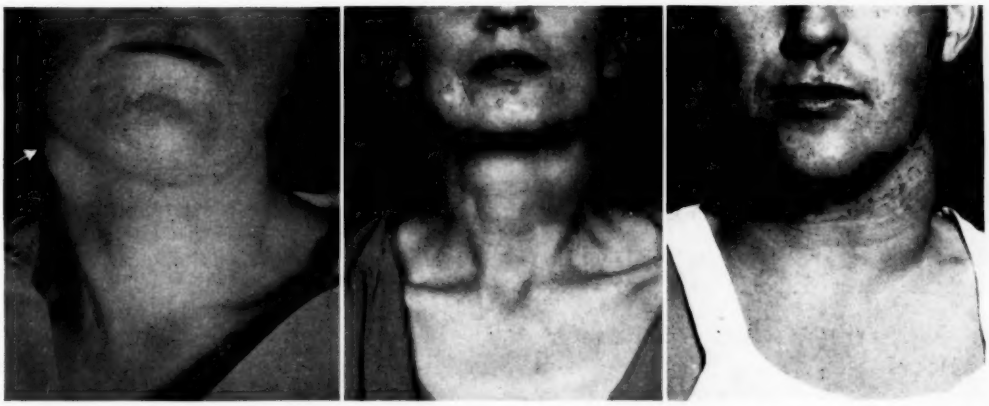

Fig. 2. All three of the patients in this illustration have carotid body tumors. Note that they are of deeper origin, enlarge inward, and originate higher than do the usual branchial cysts.

Fig. 3. This illustration shows a patient with a discrete lateral aberrant thyroid tumor. Note that similar to carotid body tumors this tumor is deep in origin, can be situated high, and is often difficult to differentiate from a carotid body tumor.

Fig. 4. This illustration shows a patient with a discrete lateral aberrant thyroid tumor which has become malignant. Note that tumors of this type can enlarge superficially and occupy the region usually occupied by branchial cysts.

in that they do not tend to enlarge upward under the jaw (Figs. 3 and 4).

Neurofibromas can, of course, occur anywhere and in our experience have occasionally been found in the neck. Arising, as they do, from nerve tissue anywhere, they have as characteristics only laterally located discreteness and movability and no typical features by which they may be distinguished from these other conditions.

While in many cases it is possible to be reasonably sure of the diagnosis of carotid body tumor, it is of importance from the point of considering preoperatively the possibility that to accomplish successfully the removal of a carotid body tumor should it prove such, one must always have in mind that ligation of the common carotid, internal and external carotid arteries may be necessary. With this possibility in mind and particularly in the light of the experience Drs. Poppen and Horrax, in the neurosurgical department of this clinic, have had with 100 cases of intracranial aneurysm, with ligation of the internal carotid in the neck in 75 cases (in 25 cases, intracranial operations were carried out), no one today should consider approaching the exploration of a carotid body tumor without preliminary compression of the carotid on the side of the neck on which the tumor is until, by pressure, complete obliteration of the carotid for a period of 10 minutes 3 times daily can be endured by the patient, with complete absence of symptoms.

In the surgical approach to any single, discrete, movable, high and laterally located tumor of the neck, we strongly urge that this preliminary carotid compression be employed. We do not believe that it is possible to be certain that any such neck tumor may not, on exposure, prove to be a carotid body tumor. If it does prove to be a carotid body tumor requiring consideration of whether or not to interrupt the carotids, it at least is some comfort to know that cerebral symptoms do or do not occur with the employment of complete carotid compression for a period of 10 minutes 3 times a day.

Compression of the carotid, worked up over a period of 7 to 8 days from 1 minute 3 times a day to 10 minutes 3 times a day as a method of determining whether or not one can ligate the common, external, and internal carotids with-

TABLE V.—PREOPERATIVE DIAGNOSIS

Carotid body tumor	9
Branchial cyst	5
Tuberculous adenitis	2
Neurofibroma	1
Lymphoblastoma	1

TABLE VI.—OPERATION

Excision with ligation of carotid vessels	6
Excision with ligation of external carotid vessel	2
Excision without ligation of carotid vessel	5
Subtotal excision without ligation of carotid vessel	1
Exploration with biopsy	2
Exploration without biopsy	2

out producing cerebral damage and a fatality, has only relative value. It is of value only when it is positive in the production of symptoms during the compression and has not in our hands proved dependable on the negative side, when symptoms are not produced by compression. We have had disaster result when such compression could be borne with no symptoms whatever and so we have placed dependence upon it solely if it is positive and symptoms are produced.

We have employed mechanical compression with the Richard light pressure apparatus and also digital compression, endeavoring to be certain of the completeness of the compression by the absence of a temporal pulse and by the complete absence of a carotid pulse when the compression has been of the digital character.

In spite of the fact that such compression could be borne without symptoms, fatalities occurred in 2 of 6 patients with carotid body tumor in whom ligation of the common, external, and internal carotid arteries was necessary in order to remove the tumors successfully. It seems probable to us from this experience that the ability of a patient to withstand carotid compression for 10 minutes 3 times a day is not a dependable indication that the common, internal, and external carotid arteries can be ligated with safety if it is necessary to do so to remove the tumor. It has value only as evidence, should the tumor be malignant, or of the type which completely surrounds the common, external, and internal carotid arteries and will require ligation of all of these vessels. A catastrophe may be expected in a high percentage of cases in which it is done.

Whether this test is or is not dependable as a test of a patient's ability to withstand common, external, and internal carotid ligation because it is impossible by means of digital pressure or mechanical pressure apparatus completely to obstruct the blood flow through the carotid or because the compression cannot be complete for a long enough period for a true test, we are unable to say. The fact, however, that 2 fatalities have occurred in patients who could stand it without symptoms does not give us confidence to ligate the carotids based upon absence of symptoms with this test.

It is to be noted in this series that 4 of these carotid body tumors were completely removed without the necessity of ligating the carotid arteries but that of these 4 cases, 1 patient died. An autopsy was obtained in this case and attention should be directed to the finding in this case of one of the dangers in the removal of these tumors even when they can be completely extirpated without the necessity of carotid ligation. Autopsy showed that although the carotid body tumor had been ablated without injury to the carotids, a sclerotic plaque had been so displaced from the intima in the manipulation of the vessel, that complete carotid thrombosis and a fatality resulted.

From the above experiences, what should be the treatment of a carotid body tumor?

The ideal treatment of carotid body tumor is complete surgical excision. The hazards of total extirpation, which derive from the necessity of ligating the carotid vessels in approximately 50 per cent of cases in which total excision is practiced, is such as to justify not employing this procedure. In an effort to formulate a rational routine of decision in this condition, there are certain factors relative to the clinical and pathologic nature of the disease, as well as to the risks of surgical excision and the effectiveness of alternative therapy, which should be kept in mind.

In approximately 75 per cent of cases the tumor is entirely asymptomatic, the presence of the cervical swelling being the only evidence of the disease. The symptoms in the remaining 25 per cent are usually trivial, and only rarely are these tumors attended by serious manifestations such as syncope. Finally, the lesion grows very slowly in most instances.

Pathologically, it is estimated that 15 to 20 per cent of carotid body tumors are malignant. Only Harrington, Clagett, and Dockerty disagree materially with this conclusion in reporting an incidence of malignancy of 50 per cent. In the material observed at the Lahey Clinic there was no proved instance of malignancy in the 18 cases.

Malignancy, when it does occur, is of low grade. Local nodes are invaded but distant metastases, suspected in a few cases, have never been verified.

Recurrence is estimated to be 7 per cent, but again Harrington, Clagett, and Dockerty reported 2 recurrences in 8 survivors following excision of malignant tumors of the carotid body, a recurrence rate of 25 per cent. It is pertinent to note that these 2 patients lived 8 and 10 years, respectively, after operation had been carried out.

The consequences of surgical excision are often serious. Phelps, Case, and Snyder, reviewing 154 proved cases, of which 148 were operated upon, reported a mortality of 24 per cent. The mortality was 30 per cent in the group of patients in whom carotid ligation was necessary. Seven per cent of those who recovered following ligation of the carotid artery had cerebral complications.

In our group of 18 cases, 17 patients complained of a swelling in the neck, while only 1 victim was unaware of the cervical tumor which was discovered during a routine physical examination. In 13 instances the tumor had gradually increased in size. Five patients had pain in the neck or face; 3 experienced hoarseness, and 1 complained of dysphagia. There was a firm tumor below the angle of the jaw in every instance. The two sides were affected almost equally, there being 8 tumors on the right, 7 on the left, and 3 were bilateral. The sexes were likewise affected equally. A thrill and bruit were definitely recorded in 3 cases and in each of these instances the tumor was compressible. Cranial nerve damage was evident in 2 patients, the hypoglossal nerve being involved in 1 case and the recurrent laryngeal nerve in another. In 2 patients the carotid body tumor had enlarged inwardly so that it bulged into the pharynx. In 1 of these the intrapharyngeal enlargement was of such extent as to interfere with swallowing and nutrition. This patient, an Army officer, had had two attempts at removal of the tumor in the Army, one by an external approach and one by an approach through the pharynx. Both were unsuccessful and the patient came to the clinic for further trial because of his inability to eat. The tumor was successfully removed by combined external and intrapharyngeal approach. With no attempt to enlarge upon the difficulty of the operation but to record the height to which these tumors can extend up under the jaw, it was finally removed only by detaching it from the base of the skull.

In the 18 cases observed at the clinic, the correct preoperative diagnosis was made in 9 instances, an accuracy of 50 per cent. The lesion was thought to be a branchial cyst in 5 cases, tuberculous adenitis in 2 patients, and a lymphoma and neurofibroma, respectively, in 2 individuals. It is interesting to record that in 1 patient in whom the clinical diagnosis of bilateral carotid body tumor was made, such a tumor was found at operation on the right and a lateral aberrant thyroid mass was removed from the left. Punch biopsy, advocated by some, is unsafe and uncertain.

Generally speaking, carotid body tumors cause no subjective complaints. Their growth is so gradual and their presence is so symptomatically benign that patients seek medical care late. In most recorded series the mean duration of symptoms before operation is 6 years and in extreme instances the tumor has been present for 35 years. The duration of symptoms in our cases varied from 3 months to 16 years.

Despite the paucity of symptoms which usually attend these tumors, a wide variety of signs and symptoms may occur. They result from stimulation or invasion of contiguous cervical structures such as the esophagus, pharynx, vagus, laryngeal, or the sympathetic nerves.

Of the 18 patients operated upon at the clinic, excision of the tumor without ligation of the carotid vessels was accomplished in 5 cases. Excision of the growth with ligation of the common, internal, and external carotid arteries was performed in 6 instances. The external carotid artery alone was interrupted in 2 individuals. The tumor was not excised in 5 patients subjected to operation because complete removal of the growth would have required carotid ligation. Subtotal resection was done in 1 instance, simple exploration in 2, and biopsy in 2 others.

There was 1 fatality in the group of 4 patients in whom the tumor was excised without sacrifice of major vessels, a percentage of 25. Death was due to thrombosis of the internal carotid artery. Two deaths occurred among the 6 patients subjected to ligation of the com-

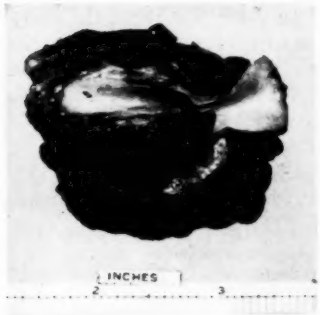

Fig. 5. This illustration and Figure 6 show carotid bodies so surrounding the external, internal, and common carotid arteries that ligature of all three vessels was necessary. Note how completely this tumor envelops all three vessels. It is in this type of carotid body tumor that removal, with the risk involved in ligating all three vessels, is unjustifiable.

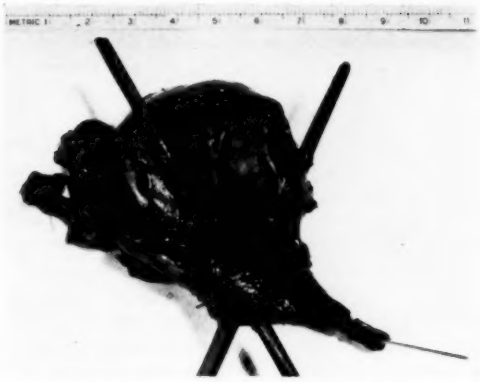

Fig. 6. See Figure 5. Note the complete involvement of the vessels (shown with rods in them).

mon, internal, and external carotid vessels, a mortality of 33 per cent. The tumors were benign in each fatal instance.

Two of the 4 patients who recovered following ligation of the carotid arteries had temporary hemiplegia with minimal residual symptoms. No mortality attended the group submitted to simple exploration, biopsy, or subtotal excision.

Eighteen patients were subjected to surgery and there were 3 deaths, a mortality of 16.6 per cent. The 3 deaths occurred in the group of 13 patients in whom the tumor was completely excised, giving a mortality for surgical extirpation of 23 per cent.

The results of alternative therapy are not encouraging. Bevan and McCarthy recorded a good response to irradiation of a carotid body tumor which was explored but not removed, and advocated conservative treatment for benign carotid body tumors whose removal would necessitate the ligation of the carotid arteries. Phelps, Case, and Snyder, contrarily, state that of 7 proved cases of carotid body tumor treated with x-rays, radium, or both, the patients failed to derive any appreciable benefit from this mode of treatment. Three patients in the clinic series were given radiation therapy with slight reduction in size of the tumor in each instance.

With all of the above facts in mind, we believe that either when the preoperative diagnosis of carotid body tumor has been correctly made or when such a tumor is demonstrated in exploring a tumor of undiagnosed origin, if the carotid body tumor is found to surround the internal, external, and common carotids completely, removal by ligation of the common carotid and internal and external carotids should be done only when biopsy demonstrates the tumor to be truly of a malignant character (Figs. 5 and 6). Even then it should be removed only when the patient has been able to work up to complete carotid compression for a period of 10 minutes 3 times a day without symptoms, and when friends and family are aware of the risks involved in such a procedure. Ligation of the common, internal, and external carotids when done should be accompanied by ligation of the internal jugular; the patient should be kept in the head down position and in an oxygen tent for a considerable period of time after operation.

When tumors of the carotid body are small, do not surround the vessels, and can be extirpated without damage to those structures, removal is justifiable. When carotid body tumors, in their enlargement, project into the pharynx by bulging the pharyngeal wall inward so that they interfere with swallowing, an attempt must be made to remove them. In neither of the 2 cases in this group in which the tumor bulged into the pharynx was there involvement of the carotids, and in both cases it was possible to extirpate the tumor without ligation of the carotid vessels.

In all cases in which the tumor, as shown in Figures 5 and 6, so surrounds the carotids that ligation of all these vessels will be necessary, the mortality of this procedure is such as not to justify its removal.

It is to be noted that our diagnoses were correct in only 50 per cent of these 18 cases. It is, therefore, evident that some of the tumors thought to be carotid body tumors will prove not to be and that some of the tumors thought not to be carotid body tumors will prove to be carotid body tumors. For these reasons, we believe that since aberrant thyroid tumors, which can closely simulate carotid body tumors, are frequently malignant and since branchial cysts can so readily be removed, and since some carotid body tumors can be demonstrated by exploration to be safely removable, all laterally located, discrete, and movable neck tumors should be explored.

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THE RÔLE OF CHRONIC THROMBOSIS OF THE PORTAL VEIN AND ITS TRIBUTARIES IN THE SYNDROME OF SPLENIC ANEMIA

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THROMBOSIS of the splenic and portal veins and splenic anemia, or Banti's syndrome, are often seen together in the same patient. Despite numerous investigations the causal interrelationships of the two processes are still not understood. The problem has assumed new importance now that some relief may be offered to patients so afflicted. Until recently the only corrective measures available for relief of portal congestion in these cases were splenectomy, omentopexy, and injection of esophageal varices by means of the esophagoscope. Splenectomy is only partially effective, since it reduces the portal blood flow by only 20 per cent (11). The value of omentopexy and injection of esophageal varices has been equivocal at best and these procedures are rarely used. Most recent efforts to combat portal obstruction are by splenorenal and portocaval anastomosis as advocated by Blakemore and Lord or by extensive gastric resection as described by Baronofsky and Wangensteen.

Splenic anemia, or Banti's syndrome, as Banti finally described it in 1910, was thought to be the result of an undiscovered infectious agent brought to the spleen by the arterial blood to produce splenomegaly and passing on to cause sclerosis in the splenic and portal veins and cirrhosis of the liver. The anemia and leucopenia were thought to result from depression of the activity of bone marrow by the toxic agent. The characteristic microscopic finding in the spleen was "fibroadenia" or fibrosis of the pulp and Malpighian bodies with retention of the normal glandlike structural appearance.

Several diseases, including Gaucher's disease (9), chronic hemolytic anemia (12), and some forms of leucemia (5,17) have been segregated as specific entities from splenic anemia while dozens of suggestions have been advanced as to the etiology and pathogenesis of the remaining large number of unsolved cases. We can divide these suggestions into 3 groups, each with numerous variations. First is the suggestion, similar to that of Banti, that the spleen is involved primarily, the changes in the veins and liver occurring secondarily as a result of liberated toxins (13). Ravenna's idea of a primary active congestion in the spleen and Pemberton and Kiernan's idea of an intrasplenic arteriovenous shunt may be included in the first group. Second is the suggestion that the spleen has a secondary rôle. This considers the process either to be a simple obstructive, congestive splenomegaly (4, 16) or to be a concomitant manifestation of hepatic disease (9). Third, the most reasonable suggestion is that splenic anemia is a symptom complex common to a miscellaneous collection of diseases in which the spleen has either a primary or a secondary rôle (6, 10). These diseases include splenoportal thrombosis, hepatic cirrhosis, and chronic infectious splenomegaly.

Several important questions arise when one attempts to decide which of these assumptions is most tenable. What is the actual cause of the splenic and portal venous disease seen in many cases of splenic anemia or Banti's syndrome? It is important to know if these venous changes are secondary to toxins or thrombogenic agents liberated by the spleen, as first postulated by Banti, or if they result from causes completely unrelated to the spleen. If

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Since this study was begun, Dr. Robertson has died.

TABLE I.—CAUSATIVE FACTORS IN CHRONIC DISEASE OF THE PORTAL VEINS (61 CASES)

No apparent cause		13
Possible or proved causes		48
Cirrhosis	19	
Abdominal operation	14	
Malignant lesion	8	
Biliary tract disease	8	
Polycythemia vera	5	
Pylephlebitis	4	
Congestive heart failure	3	
Chronic pancreatitis	2	
Syphilis	2	
Trauma	2	
Chronic hemolytic jaundice	1	
Duodenal ulcer	1	
Ingested phenol	1	
Thyroidectomy	1	
Omphalitis in infancy	1	
Subacute bacterial endocarditis	1	
Atherosclerosis	1	
Total	74*	
Total cases		61

*In several cases two or more possible causes for involvement of the portal veins were present.

the causes are unrelated to the spleen, does the obstruction and congestion caused by chronic splenic and portal thrombosis produce the splenomegaly? For comparison, does the portal congestion encountered in cases of hepatic cirrhosis cause splenomegaly or does pure chronic passive congestion encountered in cases of prolonged heart failure cause splenomegaly? Finally, what is the incidence of portal congestion and what are its causes in cases in which the clinical diagnosis is splenic anemia?

METHOD

In an effort to answer these questions we have made a clinical and pathologic analysis of data on several groups of cases in which necropsy was performed. There were 61 unselected cases of chronic disease of the splenic or common portal vein in which the disease had lasted 6 months or longer (62 per cent were males; age range, 17 to 82 years). One hundred consecutive cases of hepatic cirrhosis (70 per cent were males; age range, 12 to 72 years) were divided into the following groups: 26 with intrahepatic portal obstruction, 25 without evidence of intrahepatic or other portal obstruction and 49 unsuitable for study owing to inadequate data or to the presence of other factors which influenced splenic size. Twenty cases of heart disease were selected in which the patient had died after 2 or more years of chronic congestive heart failure (males, 65 per cent; age range, 17 to 76 years). There were 30 cases with the clinical diagnosis of splenic anemia which after pathologic study still met all the general criteria of Banti (males, 67 per cent; age range, 10 to 62 years).

RESULTS AND COMMENT

Pathology of chronic disease of the portal veins. No clinical or pathologic evidence was found to support the thesis of Banti that chronic disease of the splenic and common portal veins is caused by toxins liberated by the spleen. Instead, we found some other positive or probable cause for the chronic venous disease in 48 of the 61 cases. In most instances the cause was unrelated to the spleen. Even in 7 of the 14 cases in which a clinical diagnosis of splenic anemia had been made a well established cause completely unassociated with the spleen could be found to account for the thrombosis.

As Table I shows, there were some cases in which more than one cause was present. For example, several patients who had cirrhosis underwent surgical exploration which involved manipulation of the portal vessels. The surgical procedures were usually for disease of the biliary tract or the appendix but in one case persistent ascites developed 2 weeks after thyroidectomy and at necropsy 2 years later typical chronic thrombotic changes were observed in the portal veins. In nearly all cases of malignant lesions the growth had invaded the veins and secondary thrombosis had occurred. In several instances of disease of the biliary tract, inflammation was actually seen to spread from the common bile duct to the portal vein. Similar spread of inflammation to the veins was seen in cases in which duodenal ulcer or pancreatitis was present. Pylephlebitis is usually thought to be a rapidly fatal disease but one of these patients survived for 11 years after an acute phase of the disease to present a classic picture of splenic anemia. Pylephlebitis is probably a more frequent cause of chronic portal venous disease than is realized. Patients who have chronic congestive heart failure usually have an associated abdominal disease which also participates in the formation of thrombi. Omphalitis in infancy may

account for many of the cases of splenic anemia appearing in early childhood.

Chronic progressive thrombosis is the most common type of pathologic change in the veins. There were 39 such cases among the 53 in which microscopic sections were available. Without going into the controversy concerning pathogenesis of clot formation these lesions were predominantly thrombotic in 28 cases and predominantly phlebitic in 11.

The thrombotic vessels displayed an extremely chronic intimal thickening. Usually eccentrically placed and constricting or obliterating the lumen, the process arose from intimal proliferation and organization of thrombi. Canaliculi and deposits of iron were usually present to indicate the thrombotic origin of this change. The media was thickened, showing a cellular response where there was overlying recent thrombosis. Muscle fibers were replaced in great part by fibrous tissue. The adventitia also was thickened by an abnormal amount of fibrous tissue and free cells. In the phlebitic group the process was predominantly inflammatory, characterized by polymorphonuclear cellular infiltration. Thrombotic clots were completely overshadowed by suppuration in 2 cases of protracted pylephlebitis. In addition there were cases depicting all degrees between almost pure thrombosis and almost pure suppuration.

Eight cases fit the often quoted descriptions (7) of phlebosclerosis and were probably the result of ancient thrombosis as evidenced by scarring of the media and adventitia, the presence of canaliculi and iron deposits and the finding in proved thrombosis of venous segments which are identical to phlebosclerosis. Although there was no substantiating evidence, in some of these cases the changes may have been the result of developmental stenosis of the portal vessels.

The technical difficulties of vascular surgery and the fertile soil for postoperative thrombosis in most of the cases are obvious. The process was usually progressive with repeated thromboses superimposed one on the other and showed various stages of organization in different segments of the veins. Furthermore, in only a few cases was the disease limited to the splenic or the common portal vein; it usually extended to involve two or more major veins or into the smaller collateral veins, obstructing them and exaggerating the portal obstruction. Incidence of venous involvement was as follows: common portal, 46 cases; splenic, 44; intrahepatic branches, 41; and mesenteric, 23.

Cavernomatous transformation was a finding associated with both the thrombotic and the phlebosclerotic vessels and was interpreted as new-formed collateral circulation. Neoplastic obstruction was seen in 5 cases. In only 1 case, that of an 82 year old man, was there a small primary atherosclerotic obstruction which involved the splenic vein. Superimposed terminal thrombosis was seen in 43 cases.

Splenomegaly in chronic splenoportal venous disease. Of the 61 cases of chronic disease of the portal veins, 44 were suitable to study the effect on the spleen of chronic venous obstruction. It was found that 73 per cent of the spleens were enlarged and the mean weight was 685 grams, even though there was an extremely wide range. For those cases in which there was splenic enlargement no cause outside of venous obstruction could be found to account for the enlargement. Associated cirrhosis or toxemia of infection could have caused it in only a few cases. Why, then, were not all the spleens enlarged? Because several factors in addition to portal congestion influenced the size of the spleen. First, the presence of old infarcts with parenchymal destruction definitely prevented enlargement in two or more cases. One of these spleens weighed 35 grams and was composed chiefly of scar tissue. Second, there tended to be less enlargement among the older patients, owing to their naturally diminished splenic function and blood flow. In 3 cases of complete splenic venous obstruction in which the patients were more than 73 years of age no collateral circulation, splenomegaly or other evidence of portal congestion developed. Third, in cases of minimal portal obstruction or well developed collateral circulation there was less enlargement. Fourth, and most important, was the duration of obstruction, for in 11 cases in which the duration could be reasonably determined to be less than 2 years the splenic

mean weight was 215 grams while in 31 cases in which obstruction had lasted for 2 years or longer, the mean weight was 910 grams.

The histologic appearance of the spleens in most of the cases was uniformly alike and was remarkably similar to that of so called splenic anemia or Banti's syndrome. Fibrosis of pulp and malpighian bodies with retention of normal architectural structure was seen in both large and small spleens. Reticuloendothelial hyperplasia and perimalpighian fibrosis were more common in the larger than in the smaller spleens.

Splenomegaly in cirrhosis and in heart failure. Cirrhosis of the liver when associated with portal congestion produces a high incidence of splenomegaly (mean splenic weight, 544 grams). This is shown in 85 per cent of the 26 cases where definite evidence of intrahepatic portal obstruction was found. Portal congestion was determined in these cases by the history of gastroesophageal hemorrhage and ascites and the presence of increased collateral circulation.

McIndoe pointed out that portal obstruction in cirrhosis results from derangement of the intrahepatic portal veins by growth of hyperplastic liver cells. The obstruction may be complete long before hepatic function is appreciably diminished, so that in some cases of mild cirrhosis there is severe portal congestion. This was true in 5 of these 26 cases. Four spleens were not enlarged in spite of distinct portal congestion and no cause was found to explain this. Contrary to Rousselot's opinion, the degree of obstruction did not always determine the degree of splenomegaly.

Histologic changes in the spleens in most of these cases were similar to, or identical with, those of chronic portal venous disease or splenic anemia.

In the 25 cases of cirrhosis in which no evidence of portal obstruction was found, the mean weight of the spleens was only 281 grams. The lack of obstruction in these cases may be explained in 20 cases by the slight degree of cirrhosis. Since severity of cirrhosis and severity of portal obstruction are usually of about the same degree it is difficult to determine whether the cirrhogenous toxins acting on splenic parenchyma or the portal obstruction plays the principal rôle in splenomegaly. Apparently, as McMichael believed, both factors play a part. Histologically these spleens usually showed the early signs characteristic of splenic anemia. Some congestion was seen, although there was no evidence of it elsewhere in the portal system. Perhaps the short duration of the disease also played a large part in the lack of marked splenomegaly.

Congestive heart failure of long standing failed to produce appreciable splenomegaly (mean splenic weight, 194 gm.), even though the histologic changes were those of marked chronic passive congestion and often simulated the picture of splenic anemia. These findings seem to indicate that chronic passive congestion *per se* is not a cause of marked splenomegaly. Perhaps in order to cause splenomegaly the portal pressure must be higher than that produced by heart failure, the congestion must be active instead of passive or there must be a high differential between portal and systemic venous pressures.

Cases in which the diagnosis was splenic anemia. In the vast majority of the 30 cases in which the clinical diagnosis of splenic anemia had been made portal congestion was present. From this group with congestion it will be seen that the surgeon may expect to find chronic venous disease in half the cases (14) and cirrhosis in almost as many (11). The 14 cases in which there was venous disease have already been described among the 61 cases in which there was chronic disease of the portal veins. Many of these 61 cases were alike both clinically and pathologically whether diagnosed splenic anemia or not. Also in the group of 100 cases in which there was hepatic cirrhosis the cases with splenomegaly were similar to the 11 cases of splenic anemia with cirrhosis. As previously shown, histologic changes in the spleen are usually the same for splenic anemia, cirrhosis, and chronic disease of the portal veins.

These findings indicate that the changes form a pattern which may appear in response to more than one cause. When cirrhosis or chronic portal venous disease is manifested clinically by splenomegaly, and by anemia and leucopenia as a result of secondary splenic overactivity, the diagnosis of splenic anemia

is often made. It is easy to make a diagnosis based on such findings whereas it would be difficult to discover the underlying process.

In 3 instances of splenic anemia there was congestion with no apparent portal obstruction. These may be examples of a unique disease. The hypothesis of Pemberton and Kiernan that the portal congestion is a result of intrasplenic, arteriovenous shunt may explain these cases. Determinations, at the time of operation, of blood flow through the spleen may provide valuable information on this hypothesis. The 2 remaining cases of splenic anemia had no portal congestion or obstruction and demonstrate something we must not forget: that the syndrome of splenic anemia may be produced in an occasional patient by a cause other than portal congestion.

CONCLUSIONS

A well established cause outside the spleen can be found to account for chronic disease of the splenic or portal veins in most cases. No evidence was found which indicated that toxins liberated by the spleen cause the venous disease.

The most common type of lesion in the veins in chronic disease of the portal vein is chronic thrombosis. In the cases of severe so called phlebosclerosis the condition is the result of ancient thrombosis. Neoplastic invasion occurs in a few instances and atherosclerosis in a rare case.

In the majority, but not all, of the cases of chronic disease of the splenoportal veins splenomegaly is associated. Several factors other than portal obstruction influence the size of the spleen in these cases. Those which tend to limit the degree of splenic enlargement are splenic infarction with parenchymal destruction, advanced age of the patient, well developed collateral circulation and brief duration of the obstruction.

Cirrhosis of the liver often produces splenomegaly and the complete clinical picture of splenic anemia, but the pure chronic passive congestion of prolonged heart failure does not produce splenomegaly.

Splenic anemia is not a specific disease entity. The syndrome can be produced by a number of diseases; the most common are chronic splenoportal thrombosis and hepatic cirrhosis.

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EARLY MOBILIZATION OF PATIENTS AFTER MAJOR SURGICAL PROCEDURES

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THE idea of early ambulation following major surgical procedures is not new. Ries in 1899 was probably the first surgeon to practice this type of treatment in the country. Leithauser and Bergo (6) in 1941 reported a series of 383 appendectomies with an average postoperative confinement to bed of 1.5 days. In 1943 Leithauser (5) reported 464 consecutive major surgical procedures (including 274 appendectomies) in which all patients were ambulated in the first 24 hours. Schafer and Dragstedt, Ashkins, and Steinhart have added further cases to the long list. There are many reports in foreign literature, one of the more noteworthy being that of Kimbarovsky whose experience covered 551 cases. Many of his patients walked from the operating table.

Since January, 1945, we have been mobilizing our private patients early after operation. Excluding hemorrhoidectomies, excision of small tumors, fractures, spine fusions cases and the like, there were, between January 1945 and March 1946, 266 unselected, consecutive cases of major surgical procedures, following which early postoperative ambulation was carried out. In an attempt to discover any advantages accruing from this method, a control series was carefully studied. This control series consisted of 221 consecutive major operations done from January, 1944 to January 1945, by the same surgeon, whose only variation in procedure after January 1, 1945 consisted in getting the patient up early. The equivalence of factors renders these two series ideal for comparison.

The 1944 group will hereafter be referred to as group I, and the early ambulation group as group II. In both groups postoperative care was well controlled, and 92.7 per cent of the patients were followed carefully for periods ranging from 9 to 21 months.

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Dr. Lin has previously been listed in the literature as David Lin-Yuen.

PROCEDURE OF EARLY AMBULATION

Our present method consists in getting patients up 24 hours after operation, irrespective of the type of operation. The patient, having a tight binder applied, is elevated to a high Fowler's position and his legs are swung over the side of the bed. He is gently supported to the standing position, and assisted to a chair a few steps away, where he sits for a period of 15 to 30 minutes. On the second postoperative day, this procedure is carried out twice. From then on, the time out of bed and the length of the walks are progressively increased. By the fifth to seventh day the patient is walking to the bathroom himself. Sutures are removed on the 5th or 6th day, and stay sutures, if any, on the 8th day, unless there is some contraindication. Patients having undergone appendectomies and thyroidectomies are sent home by the 5th to 7th day, while all others are usually home 7 to 10 days after operation.

Apart from exceptional cases, no patient is allowed to be catheterized. If there is difficulty in urination and defecation, the patient is allowed up as often as is necessary even on the day of operation.

The rest of the postoperative care needs no detailed discussion here. Careful observations of the pulse and blood pressure, and routine rebreathing and postoperative exercises are carried out while the patient remains in bed. He is urged to cough, even though the act may cause discomfort. Fluid and electrolyte balance is well controlled.

A summary of the data on all 487 cases of both groups is presented in Table I. In evaluating the results of this study, both averages and medians are calculated. Because a few values fall far outside the normal distribution curve thus distorting average values, standard deviation from the median is used throughout.

TABLE I.—SUMMARY OF CLINICAL DATA ON 487 MAJOR OPERATIONS

	Average entire series 487 cases	Group I. Late ambulation 221 cases		Group II. Early ambulation 266 cases	
		Average	Median $\pm \sigma$	Average	Median $\pm \sigma$
Average age of patients	43.4	44.6	$46 \pm \sigma 16.5$	40.9	$47 \pm \sigma 14.4$
Maximum postoperative rise in temperature	1.4 C	1.4 C	$1.4 \pm \sigma .57$ C	1.3 C	$1.4 \pm \sigma .54$ C
Postoperative day for maximum temperature rise	1.4	1.4	$1 \pm \sigma 1.9$	1.3	$1 \pm \sigma 2.4$
Postoperative day for return of temperature to normal	4.9	6.2	$4 \pm \sigma 5.9$	4.6	$4 \pm \sigma 2.8$
Postoperative day for ambulation	4.3	9.5	$10 \pm \sigma 5.8$	2.3	$2 \pm \sigma 3.5$
Postoperative day for discharge	12.8	15.4	$13 \pm \sigma 9.9$	11.1	$11 \pm \sigma 7.7$
Complications	28 (5.7%)	18 (8.1%)*		10 (3.8%)*	
Deaths	20 (4.1%)	10 (4.5%)		10 (3.7%)	

*Standard error of the difference 2.3%: actual difference 4.3%.

Referring to Table I, one finds the median age of group I is $46 \pm \delta$[1]16.5 years, while that of group II $47 \pm \delta 14.4$ years. The 2 groups studied are thus similar in age.

In like manner, it is shown that the figures for maximum postoperative rise in temperature, postoperative day for maximum rise in temperature, and postoperative day for return of temperature to normal are statistically the same in both groups.

Now, in examining the median day for ambulation it is $10 \pm \delta 5.8$ days for group I, and $2 \pm \delta 3.5$ days for group II. The difference in the two groups might not seem statistically significant. However, reference to Figure 1 shows that group II does not form the normal distribution curve but half of such a curve, the peak being along the 1st postoperative day. With group I, the peak of the distribution curve is along the 10th postoperative day. Considering this skew, the difference in the 2

[1] δ = Standard deviation of

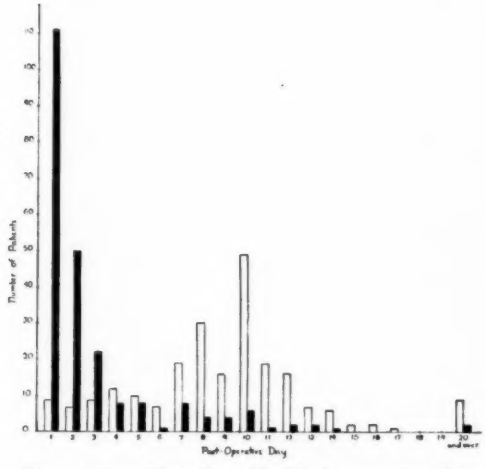

Fig. 1. Group I is indicated by blank area, group II by black area.

TABLE II.—CLASSIFICATION OF OPERATIVE PROCEDURES

Operation	Group I	Group II	Total
Herniorrhaphy			
Inguinal, unilateral	51 (2R)	63 (4R)	114
Inguinal, bilateral	16 (1R)	22 (3 with one side R)	38
Femoral	4	3 (1R)	7
Ventral	11	8 (1R)	19
Lumbar	1 (R)	0	1
			179
Cholecystectomies	26 (2)*	61 (2)	87 (4)
Hysterectomies	31 (5)	26 (6)	57 (11)
Appendectomies	28	14	42
Gastroduodenal operations	18 (9)	23 (9)	41 (18)
Lower gastrointestinal operations	14 (6)	21 (13)	35 (19)
Thyroidectomies	11	15	26
Mastectomies	7 (7)	4 (4)	11 (11)
Gynecological repairs	3	6	9
	221 (29)	266 (34)	487 (63)

*Numbers in brackets denote malignancies.
R—Recurrent.

TABLE III.

Group I—January 1944 to January 1945

No.	Age years	Diagnosis	Operation	Complication	Post-operative day	Result
1	50	Pericecal abscess	Drainage of pericecal abscess	Cecal, fistula	11	Healed, subsequent herniation
2	72	Chronic cholecystitis with stones	Cholecystectomy with drainage	Atelectasis	3	Resolved
3	55	Carcinoma of ascending colon	Resection of ascending colon	Bronchopneumonia, abdominal abscess	35	Resolved
4	51	Goiter, toxic diffuse	Subtotal thyroidectomy	Coronary occlusion	3	Resolved
5	54	Bilateral inguinal hernia	Bilateral inguinal hernioplasty	Hematoma into scrotum with cellulitis	1	Resolved
6	57	Chronic cholecystitis with stones	Cholecystectomy with drainage	Disc atelectasis	13	Resolved
7	73	Carcinoma of stomach, inoperable	Anterior gastroenterostomy	Disc atelectasis	10	Resolved
8	27	Incarcerated inguinal hernia	Inguinal hernioplasty	Thrombophlebitis	12	Resolved
9	68	Penetrating duodenal ulcer	Gastric resection	Thrombophlebitis	10	Resolved
10	54	Carcinoma transverse colon and rectum	Abdominoperineal resection	Atelectasis, ileus Empyema of gall bladder	15	Resolved; gall bladder drained
11	44	Chronic cholecystitis with stones	Cholecystectomy	Atelectasis	3	Resolved
12	62	Carcinoma of stomach	Gastric resection	Wound infection	7	Resolved
13	50	Carcinoma of cecum with metastasis	Resection of cecum	Wound infection	6	Resolved
14	70	Acute cholecystitis abscess of pancreas	Laparotomy	Thrombophlebitis	44	Resolved
15	43	Incisional hernia	Herniorraphy	Bronchopneumonia	1	Resolved
16	58	Incisional hernia	Herniorraphy	Thrombophlebitis	10	Resolved
17	48	Myoma uteri	Hysterectomy	Abscess of abdominal wall	10	Subsequent incision and drainage required
18	61	Indirect inguinal hernia	Herniorraphy	Atelectasis	2	Resolved

Group II January 1945 to March 1946

1	36	Left inguinal hernia	Inguinal hernioplasty	Exacerbation of chronic nephritis	10	Improved
2	72	Carcinoma of descending colon	Resection of descending colon; end-to-end anastomosis	Fecal fistula	15	Closed
3	59	Chronic appendicitis	Appendectomy; lysis of adhesions	Bronchopneumonia	1	Resolved
4	68	Chronic cholecystitis with stones	Cholecystectomy with drainage	Disc atelectasis	4	Resolved
5	73	Carcinoma of rectosigmoid	Resection of rectosigmoid with end-to-end anastomosis	Wound infection	7	Resolved
6	53	Peptic ulcer	Gastroenterostomy	Renal failure	8	Improved
7	47	Myoma uterus	Hysterectomy	Wound infection	8	Resolved
8	69	Carcinoma of rectum	Abdominoperineal resection	Bronchopneumonia	4	Resolved
9	70	Sarcoma of uterus	Panhysterectomy	Coronary occlusion	2	Improved
10	51	Ventral incisional hernia with fecal fistula	Ventral hernioplasty	Wound infection	7	Resolved

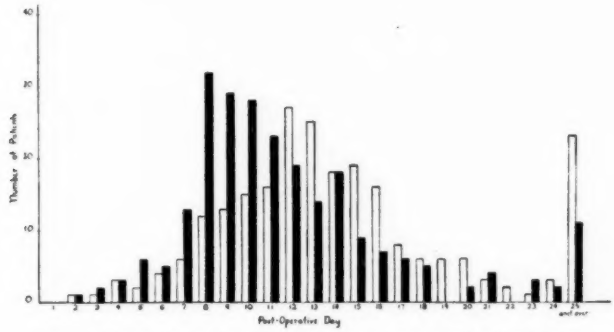

Fig. 2. Group I is indicated by blank area, group II by black area.

groups is greater than could occur by chance, and bears out the claim of early rising for group II.

The median day for discharge for group I was 13th postoperative day $\pm \delta$ 9.9 days, that of group II the 11th day $\pm \delta$ 7.7 days. This difference could have occurred by chance, but Figure 2 shows the trend towards earlier discharge for group II. The peaks of the curves show a difference of 4 days in day of discharge.

In group I there were 18 complications or 8.5 per cent, and in group II, 10 complications or 3.8 per cent. The standard error of the difference is 2.3, while the actual difference was 4.7 per cent. This difference could have occurred only 1 in 20 times on the basis of chance, and is, therefore significant.

TYPES OF OPERATIONS

Table II indicates the types of operations performed, with the number of each. All these cases were done under a combined anesthesia of avertin, gas-oxygen-ether.

Hernia. In the repair of all hernias, inguinal, femoral, ventral or lumbar, an all silk technique is employed. The wound edges are blocked off with towels as soon as the skin incision is made so as to prevent contamination. The Halsted type of repair is used in both direct and indirect inguinal hernias. Both sides of a bilateral hernia are repaired at one sitting.

Biliary surgery. In cases of biliary tract surgery the approach is through a right paracostal or Kocher type of incision. The wounds are closed with chromic catgut for peritoneum and fascia, with interrupted silk for the skin, and silver wire as stay sutures. Drainage is maintained for 48 hours.

Laparotomies. Gynecological repairs, thyroidectomies, radical mastectomies complete the list.

As indicated in Table II, there were 63 operations for malignant conditions, 34 of which occurred in group II.

Early postoperative complications. Tables III and IIIA reveal not a single case of dehiscence or eventration, either in group I or group II. While wound infections numbered 3 in each group, none of these was among the 179 hernioplasties. There were 4 cases of thrombophlebitis in group I, and none among the early ambulation group. Pulmonary complications were 8 in group I as compared to 3 in group II. Coronary occlusion occurred once in each group. Renal complications were more prevalent in group II. One was an exacerbation of pre-existing chronic nephritis after her-

TABLE IIIA.—SUMMARY OF EARLY POSTOPERATIVE COMPLICATIONS

Complications	Group I	Group II	Total
Coronary occlusion	1	1	2
Renal disturbances	0	2	2
Bronchopneumonia	2	2	4
Disc atelectasis	6	1	7
Thrombophlebitis	4	0	4
Hematoma in wound	1	0	1
Wound infection	3	3	6
Fecal fistula	1	1	2
	18	10	28

TABLE IV.—SUMMARY OF 20 POSTOPERATIVE DEATHS

No.	Age	Diagnosis	Operation	Post-operative day ambulation	Complication	Cause of death	Postoperative day of death
			Group I				
1	35	Ulcerative colitis	Emergency laparotomy; cecostomy, aspiration gall bladder	22	Bronchopneumonia	Same	46
2	50	Carcinoma of pancreas; metastases to liver	Cholecystojejunostomy	8	Pulmonary congestion	Same	18
3	56	Acute cholecystitis and cholelithiasis	Cholecystectomy	—	Shock	Same	2
4	57	Inoperable carcinoma of stomach	Laparotomy	3	General deterioration	Same	45
5	68	Inoperable carcinoma of stomach	Laparotomy	8	Carcinomatosis	Same	25
6	77	Cystadenocarcinoma of ovary	Excision of ovary	—	Carcinomatosis	Same	3
7	57	Recurrent carcinoma of cecum	Ileocolostomy	—	Carcinomatosis	Same	35
8	56	Carcinoma of head of pancreas	Cholecystojejunostomy	—	Cerebral hemorrhage	Same	24
9	75	Recurrent direct inguinal hernia	Herniorrhaphy	—	Acute myocardial infarction	Same	5
10	77	Mesenteric thrombosis	Laparotomy		Auricular fibrillation	Same	At operation
			Group II				
1	67	Periappendiceal abscess with peritonitis	Drainage of periappendiceal abscess	—	Bronchopneumonia, Ileus	Same	18
2	77	Carcinoma of gall bladder metastases to liver	Resection portion of right lobe of liver and of gall bladder	1	Carcinomatosis	Same	115
3	77	Strangulated inguinal hernia	Reduction of strangulated hernia and hernioplasty	—	Cardiac failure, pneumonia	Same	2
4	74	Carcinoma of stomach	Gastrectomy	—	Renal failure	Same	5
5	53	Metastatic carcinoma of liver	Laparotomy	6	General deterioration	Same	44
6	48	Perforated marginal (jejunal) ulcer	Dismantling gastroenterostomy and jejunal resection	14	Renal failure	Same	21
7	61	Acute cholecystitis with abscess	Drainage of abscess of gall bladder	—	Pulmonary congestion	Same	3
8	40	Thrombocytopenic purpura	Splenectomy	—	Shock	Same	0
9	53	Duodenal ulcer	Gastrectomy	—	Shock	Same	0
10	64	Perforated duodenal ulcer	Gastrectomy	—	Embolism	Same	5

nioplasty, the other was a temporary renal failure after gastroenterostomy.

Deaths. There were 10 postoperative deaths in each group. Tables IV and IVA give a detailed account and summary report of these deaths. It will be noted that 6 patients in group I and 7 in group II died before getting out of bed. In no case can it be shown that early rising would have prevented death. In all 20 cases of both groups, 3 patients died from operative shock, 3 from cardiac failure, 2 from pulmonary congestion, 1 from embolism, 6 from carcinomatosis, and 1 from cerebral hæmorrhage. Thus 50 per cent of the deaths can be classified as cardiovascular deaths.

FOLLOW-UP

Of the 487 cases done, 467 patients were, discharged alive. There were 17 cases (or 3.7%) that could not be followed. The remaining patients were examined and data carefully recorded. Follow-up visits were slated for every 3 to 6 months. Table I shows in detail the follow-up record—it is evident that 417 out of 450 patients (or 92.7%) had a follow-up of 6 months or more, and 289 (or 64.2%) had a follow-up of 9 months or more.

TABLE IVA.—SUMMARY OF CAUSES OF DEATH

	Group I	Group II
Operative shock	1	2
Cardiac failure	2	1
Renal failure	0	2
Bronchopneumonia	1	1
Pulmonary congestion	1	1
Embolism	0	1
Carcinomatosis	4	2
Cerebral hemorrhage	1	0
	10	10

TABLE V.—FOLLOW-UP ANALYSIS OF 450 CASES*

Period of follow-up months	Group I	Group II	Total
12–21	127	66	193
9	30	66	96
6	37	91	128
Less than 6	11	22	33
	205	245	450
Late complications			
Incisional hernia	4	2†	6
Recurrence after hernioplasty	2	1‡	3
Recurrence of hydrocoele	1	0	1
Keloid formation	2	2	4
Latent stitch abscess	1	2	3
Tenderness in scar	2	2	4

*20 deaths, and 17 cases (or 3.7%) with no follow-up.
†Small herniations at site of the drain.
‡Preoperative diagnosis: recurrent inguinal hernia.

Though a minimum period of 1 year should constitute a fair follow-up for herniation to form or reform, we believe that most hernias occur or recur within the first 6 months after operation. It will be noted that in group II, all hernioplasties were ambulated in the first 24 hours. This group included 4 unilateral recurrent inguinals, 3 one-sided recurrences in bilateral inguinals, 1 recurrent femoral, and 1 recurrent ventral. In follow-up there was only 1 recurrence in a previously recurrent inguinal hernia as contrasted with 2 recurrences in group I.

Also 4 cases of incisional hernia in group I and 2 small herniations in group II were present at the site of drains.

There were no significant differences in the cosmetic and functional results of the two groups.

EVALUATION OF EARLY AMBULATION

Physiologically, early ambulation appears to be sound. Leithauser (5) showed that prolonged recumbency reduces the vital capacity of the lungs, due to reflex inhibition of the diaphragm following abdominal procedures. Churchill and McNeil have expanded upon this point. Standing patients on their feet helps pull down the diaphragm, giving better aeration to the lungs. Deep breathing and forced coughing rid the bronchioles of mucous plugs.

Dock believes that the bed pressure on partially filled vessels may cause endothelial changes that give rise to phlebothrombosis, with extension from peripheral radicals to deeper vessels of the pelvis, with possible dislodgement of fatal emboli.

Early exercise promotes better circulation, and must increase the blood supply to all parts of the operative site. The muscles do not atrophy from prolonged stay in bed, and the patient preserves his muscular tone and development.

From the patient's standpoint then, his feeling of well-being is restored earlier. He does not feel he has been an invalid. He is happy not to have been catheterized and not to have had to use the bedpan. He may have been fearful at the prospect of early rising, so soon after operation, but became confident after the first trial. Many look back on the idea of early ambulation with approval.

One patient, with repair of a double hernia, decided that since he was discharged early, he could return to work. At the end of 2 weeks, he was doing his regular job, which included lifting 60 to 70 pounds many times a day. He suddenly realized that he would be ineligible for compensation if he continued, so after 3 weeks of heavy labor, he stopped work. No ill effects were demonstrable in this patient at subsequent follow-up, which has covered a period of 1 year.

With the great shortage in hospital beds today, the reduction by even 4 or 5 days of hospitalization for each patient is a tremendous

factor in solving this shortage. There is more rapid turnover in cases, which is of interest to the resident staff. The acute shortage of nursing personnel has been alleviated somewhat by early mobilization of patients, who are able to take care of themselves early in their convalescence. In the case of those who do not possess hospital insurance, the economic advantage of a shortened hospital stay is quite obvious.

SUMMARY

1. Two groups of cases are reported: group I, a control group of 221 consecutive major operations done in 1944 on patients, who were ambulated late; group II, a series of 266 consecutive major operations done by the same surgeon in 1945 on patients, who were mobilized 24 hours after operation.

2. There was no significant difference in the postoperative temperature rise nor in the time required for remission of fever.

3. The peak of the ambulation curve for group II was along postoperative day 1, and for group I, postoperative day 10.

4. Patients in the early ambulation group are discharged some 4 days earlier than those of the late group.

5. There were 18 complications (8.5%) in group I, and 10 complications (3.8%) in group II. The difference is statistically significant.

6. There were 8 pulmonary complications in group I and 3 in group II.

7. There were 5 cardiovascular complications in group I, and 1 in group II. No case of thrombophlebitis occurred in the early ambulation group.

8. Not a single case of dehiscence occurred in either group, although in group II, debilitated patients were subjected to the same early rising routine as the more robust.

9. No significant difference was present in the causes of deaths in either group.

10. There were 97.3 per cent of cases with follow-up, of which 92.7 per cent were followed from 9 to 21 months.

11. There were 4 cases of incisional hernia in 205 patients in group I or 1.9 per cent; 2 instances in 245 patients in group II or 0.8 per cent. There were 2 cases of recurrences of inguinal hernia in group I, and 1 case in group II.

12. The physiological, psychological, and economic advantages of early ambulation are considered.

CONCLUSION

On the basis of this study of 487 unselected, consecutive major surgical cases, early ambulation after operation seems to have no ill effects on the patient. On the other hand, early and late complications are visibly lessened in the early rising group.

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LUNG MOBILIZATION

Its Indications in the Management of the "Captive" Lung

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THE extensive collective experience which surgeons had as a result of the recent war made possible many advances in traumatic surgery. Above and beyond the general advances made in such fields as chemotherapy or blood replacement, spectacular achievements can be pointed out in specialized practices. In thoracic surgery, the light shed on reflex changes incident to chest trauma (1, 2, 3), the bold application of decortication or lung mobilization in organizing hemothorax and hemothoracic empyema may be pointed out among others, as achievements of high order in this specialized field (3, 6, 8, 11, 12).

The application to civilian practice of lessons learned from military experience is not always easy and certainly thoracic trauma during peace time is not very common, but, to stop by simply saying that decortication is indicated in organizing hemothorax would be to miss a much more fundamental principle for which lung mobilization was actually used and possibly to exclude from the surgeon's repertoire a useful approach to problems of chest disease. It seems justifiable therefore to consider the maneuver of lung mobilization at thoracotomy in light of its basic indication.

When critically surveyed, decortication or lung mobilization was used in military surgery to provide for the expansion and consequent filling of the hemithorax by useful lung. In organizing hemothorax this was accomplished by removing the "coat" or "peel" of organizing fibrin which closely invested the visceral pleura and by this mechanical means effectively prevented any increase in pulmonary volume. The applicability of this basic principle to civilian type surgery seems to justify elaboration.

The total number of factors which may influence or to some extent control pulmonary re-expansion after collapse is in all probability great. However, from a practical viewpoint it is believed that they may be subjected to reasonable classification (4). If considered in the abstract, this problem is very complicated but basically involves an increase in volume of the collapsed lung. To accomplish this, three circumstances must obtain: (1) Some substance (air) must enter the lung to provide this bulk; (2) the lung itself must be capable of expansion; and (3) the remaining content of the hemithorax (that content which made up the difference between the volume of the collapsed lung and the volume of the hemithorax) must suffer a decrease in volume corresponding to the first proposition above.

This set of circumstances is applicable in the most commonly seen problem of pulmonary re-expansion, namely, massive collapse. In atelectasis, relief of the bronchial obstruction by permitting the ingress of air satisfies the first proposition, the lung is usually intrinsically capable of expansion, fulfilling the second proposition, and the remaining pleural content is correspondingly displaceable since it is made up mainly of a shifted mediastinum and elevated hemidiaphragm. A situation easier to understand than the example of atelectasis is, of course, the common and pertinent one of failure of postthoracotomy re-expansion of the lung or postlobectomy re-expansion of the remaining lobe or lobes. Therefore, in attempting to arrive at a more practical consideration of the problem and avoid a tedious discussion of theoretical variants, the following presentation makes the basic premise that the chest wall is functionally intact.

Putting the three basic requirements for pulmonary re-expansion into other words, permits the elaboration of the following scheme of influential factors:

1. Bronchial
   - Defects of the large or segmental branches
   - Defects of the smaller branches

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II. Pulmonary
  Defects of the alveoli
  Defects of the interstitial tissues
  Defects of combined alveolar and interstitial types
III. Pleural. The presence within the pleural space of:
  Air
    1. Simple mantle without tension
    2. Due to a bronchopleural fistula
      a. Nonvalvular defect without tension pneumothorax
      b. Valvular leak with tension pneumothorax
  Fluid serous, sanguineous, purulent, etc.
  Fibrin
  Pleural investments or distorting pleural symphyses

The bronchial factors are rather obvious and can be brought under the general term of obstruction. Such obstruction may be due to retained secretions, or, in postlobectomy lobar collapse, to edema spreading from an adjacent line of bronchial resection, or even to distortion of the bronchus, etc. (5, 7, 10).

The defects of the smaller ramifications of the bronchial tree would also include retained secretions. Their presence in a sufficiently wide area of the bronchial tree to be of significance in a postthoracotomy patient could, for example, result from the application of positive pressure by the anesthetist without adequate prior tracheobronchial toilette. The possibility that tracheobronchial secretions may actually be driven beyond easy reach by poorly managed positive pressure anesthesia is probably not as widely appreciated as it should be.

The pulmonary factors are usually not encountered in a recently thoracotomized patient, but do assume importance in long-standing collapse. Edema, exudate or blood may, of course, fill the alveoli, rendering them airless and heavy and, thereby, prevent adequate expansion (11). Extensive retention of lipiodol may possibly be operative in this manner (5). Because of prior disease or long-standing collapse under a progressive type of pleural exudate, the interstitial tissues may be the site of such extensive fibrosis that they will not permit adequate increases in pulmonary volume (12).

The pleural factors may, of course, be varied and many. They all embody, however, the idea of preventing increases in pulmonary volume because of their presence and if they are the only factors preventing lung expansion, a direct approach for their correction is not only possible but is usually necessary. Just as bronchoscopy may disclose or remove the bronchial factor, so thoracentesis can demonstrate and correct most of the pleural factors.

The presence of a simple mantle of air provides a self-correcting situation because it can be expected to undergo absorption and disappear eventually. A tension pneumothorax, however, is a much less benign situation, but again manageable by well established routine. Certainly a lung cannot expand under an undecompressed tension pneumothorax, but the recognition of this condition poses no unusual problem. The presence of a nonvalvular leak of small size may not prevent eventual lung expansion, but will certainly delay it. A tension pneumothorax provides for progressive lung collapse, whereas the nonvalvular leak not only maintains an adverse pleural factor (pneumothorax), but also interferes materially with the efficiency of pulmonary inflation. Even though neutralization of these situations may tax the surgeon's ingenuity to the utmost, such discussion is not within the scope of this paper.

Pleural fluid as a factor probably needs no comment except to point out its significance in being usually associated with some more fundamental pathologic process.

The presence of fibrin or pleural investments or deforming pleural symphyses strikes a new note in this problem in direct application of the lessons learned in the recent war. The presence of fibrin prevents expansion by its bulk, but if it be removed, the remaining and probably constantly concomittant pleural coat of organized fibrin will still prevent or greatly delay expansion. This situation provides a lung that may be entirely capable of expanding except for the fact that it is retained or held captive, mechanically, in the collapsed position, by a restricting fibrous investment. That minor degrees of this process in the form of distorting pleural symphyses may greatly delay expansion, has previously been pointed out (9).

Returning now to review this problem in its entirety, bronchoscopy, clinical evaluation, and thoracentesis provide the essential diagnostic as well as therapeutic approaches. Bronchoscopy should reveal and/or eliminate the bronchial factor. Clinical evaluation at least, should reasonably, disclose and indicate treatment for a pulmonary factor. Obviously such appraisal may of necessity amount to an extensive investigation, but in the recently thoracotomized patient the status of the lung should be well known, either because of the preoperative studies or the information obtained at operation or both. Thoracentesis should permit accurate recognition of most of the possible pleural factors, can correct many of them, and should be able to point to proper treatment in all.

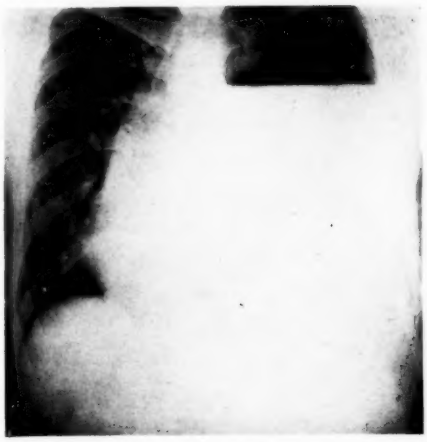
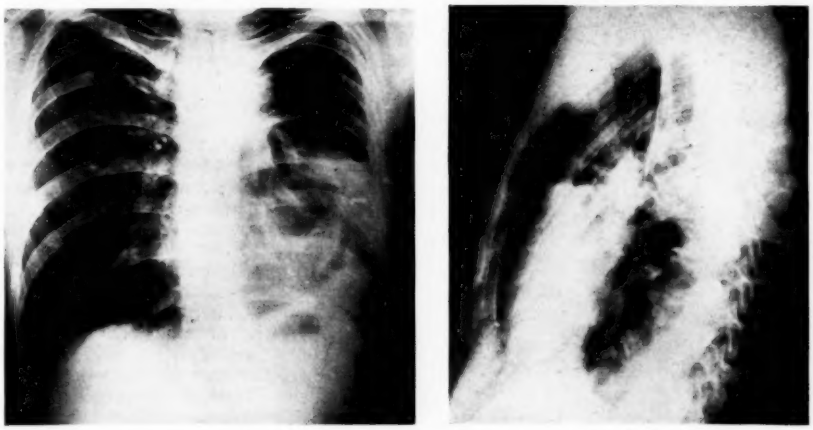


Fig. 1. Case 1. Admission roentgenogram, indicating the extensive left hydropneumothorax which totally covers the lung and greatly displaces the mediastinum toward the right.

A pleural factor not reducible by thoracentesis, such as fibrin, or the lung which refuses to expand after removal or neutralization of the pleural factor, requires thoracotomy for its correction, subservient, of course, to the previously mentioned diagnostic steps by which important and basic etiologic considerations should be elucidated.

It is believed, therefore, that if a patent and normal bronchial tree is present in a lung that has no intrinsic cause for nonexpansibility yet fails to expand after elimination or neutralization of the pleural factor, the diagnosis of a captive lung or a lung that is retained mechanically in this state of collapse can be made with reasonable certainty and thoracotomy for its



a b

Fig. 2. Case 1. a, Posteroanterior roentgenogram taken before thoracotomy and decortication. Although the mediastinum has returned to a more normal position, the lung has failed to re-expand significantly, particularly in its apical portion. The evidence of basal loculation is noted and what apparent filling of the hemithorax has occurred is actually due to continued and severe contraction of the chest wall. b, Lateral roentgenogram of same date as a.

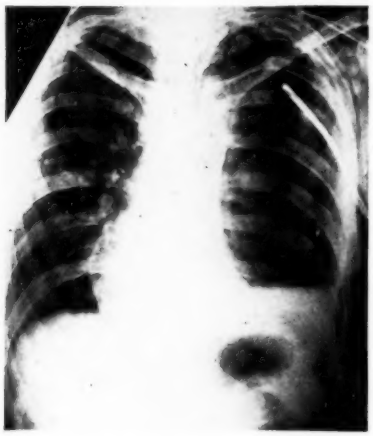


Fig. 3. Case 1. Bedside roentgenogram taken 48 hours after decortication. The basal fluid level is the result of accidental occlusion of the posterior intercostal drain. The lung is, however, virtually completely re-expanded.

correction is justified, if such prompt re-expansion is not contraindicated on etiologic grounds.

The following case reports will serve as illustrations of the principles which have been discussed.

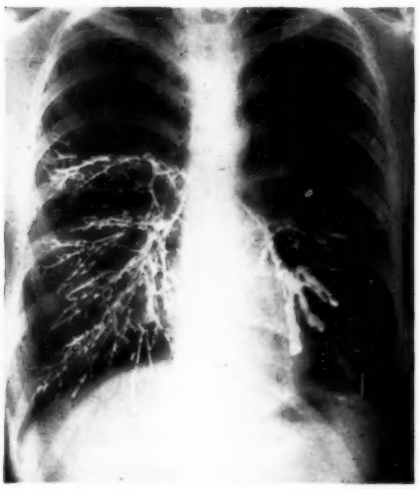


Fig. 5. Case 2. Bronchogram illustrating the extent of bronchiectasis. Additional projections and a more complete left-sided filling were used to clearly indicate the extent of involvement of the upper lobe.

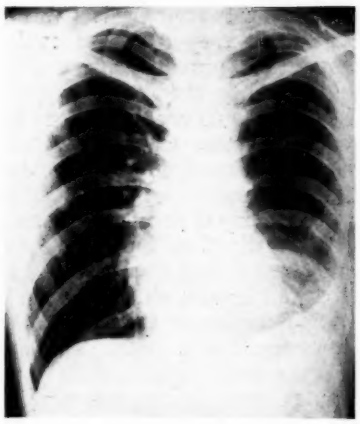


Fig. 4. Case 1. Roentgenogram showing final result. An intercostal incision was used without rib resection. The left hemithorax is approaching normal volume and a good functional result was obtained even though lateral "tenting" of the hemidiaphragm is present.

CASE 1. G.R.B., male, white, aged 30 years, was referred for treatment with the following history. On November 19, 1945, while sitting at the breakfast table, he developed a sudden sharp pain in the epigastrium, accompanied by shortness of breath. He was treated by bedrest and given symptomatic care for 4 or 5 days, when he was hospitalized. An effusion in the left chest was recognized and 200 cubic centimeters of air and 200 cubic centimeters of bloody fluid were aspirated, providing some relief. Because there had been no noticeable improvement in the condition of the chest even though symptoms had abated, he was referred for definitive care on December 29, 1945.

The admission x-ray film revealed an extensive hydropneumothorax on the left (Fig. 1). This was decompressed and the pleura emptied, requiring partial air replacement for control of negative pressure symptoms. The fluid was thin, bloody, and sterile. No acid-fast bacilli were found in the pleural fluid nor in the gastric content. There was no sputum and no history of lung disease. Bronchoscopic examination revealed a dry and patent bronchial tree. The pleura was repeatedly aspirated, removing air and any reaccumulating fluid (which was negligible in amount), but no indication of pulmonary re-expansion could be detected. Careful manometric readings of intrapleural pressures gave no indication of a bronchopleural fistula. Instead, contracture of the left chest became more pronounced. On the basis of these findings, the lung was considered to be "captive," probably on the basis of an investing pleural "coat" or binding pleural adhesions (Fig. 2a and b).

On January 28, 1946 a thoracotomy was performed. The lung was found to be in a state of virtually complete collapse and covered by a "peel" typical of or-

Fig. 6. Case 2. Bedside roentgenogram of July 27, 1946. The collapsed lobe is clearly seen, as is the mediastinal shift and elevation of the left hemidiaphragm. A fluid level is present, as well as the suggestion of a mass partially filling the pleural space.

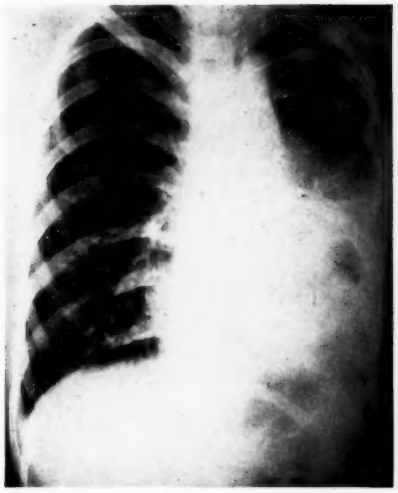


Fig. 7. Case 2. Bedside roentgenogram of August 3, 1946. There has apparently been some readjustment of the mediastinum toward its normal position, but no increase in volume of the remaining lung. Fluid and the probable intrapleural mass persist despite all efforts at emptying the pleura.

ganizing hemothorax some 2 millimeters in thickness. Decortication was surprisingly easy and lung expansion full and prompt. His postoperative course was uneventful and he was discharged in a state of full functional capacity (Figs. 3 and 4). The site of leak from the lung which had resulted in the spontaneous hemopneumothorax was not recognized at the time of operation.

CASE 2. R.M., male, white, aged 21 years, was admitted on June 12, 1946 for treatment of bronchiectasis, for which he had received a medical discharge from military service. A productive cough had been present since 1940, which symptom had appeared following an attack of "influenza." Two bouts of hemorrhage had been experienced.

After appropriate study and a suitable period of preoperative preparation (Fig. 5), the left lower lobe and the lingular portion of the upper lobe were resected by individual ligation technique without difficulty on July 25, 1946. Anterior and posterior intercostal drains connected to water seals were employed. The remaining lung inflated in an entirely satisfactory manner prior to closure. The postoperative reaction was minimal and no concern over his welfare was felt until the bedside roentgenogram on July 27, 1946 (Fig. 6) was seen, despite the unfortunate emptying of one of the water seals shortly after operation.

Renewed efforts in order to insure adequate evacuation of the pleura were made, which included aspiration of the chest, breathing exercises, forced coughing and continuous pleural decompression. Suction on the intercostal catheter was poorly tolerated because it soon produced acute thoracic discomfort. Sputum was being adequately raised. The fluid obtained from the left pleural space was sterile on culture, but was viscid and serofibrinous in character. At bronchoscopy, the bronchial tree was relatively dry, the upper lobe orifice was clearly patent, there being no rotation or encroachment by edema to any significant degree. A curved vertebrated suction tip could be easily introduced into the upper lobe bronchus. No secretions were obtained by this maneuver. Throughout this entire time the patient's clinical course was very favorable, despite persistent collapse of the remaining lung (Fig. 7). Thus, since the bronchial factor was presumably eliminated, there was no known or demonstrable pulmonary factor, no bronchopleural fistula could be demonstrated and evacuation of the pleural content, which was as complete as could be tolerated by the patient (checked by fluoroscopy) failed totally in promoting re-expansion of this lobe, the diagnosis of a "captive" lung or a lung retained in the collapsed position, probably by fibrin, seemed justified.

Accordingly, on August 6, 1946 the thoracotomy wound was reopened. The pleural space was found to contain some residual fluid, but a large mass of fibrin comprised most of the volume therein and closely invested the lung which lay totally collapsed in the gutter posteriorly. After manual removal of this fibrin mass, a peripleural coat of organized fibrin was recognized and decortication undertaken and carried out fairly completely, although its age (only 12 days) rendered it thin enough that some shredding occurred. This procedure was carried out over the

Fig. 8. Case 2. Bedside roentgenogram 48 hours following second thoracotomy. Immediate and very gratifying re-expansion has occurred.

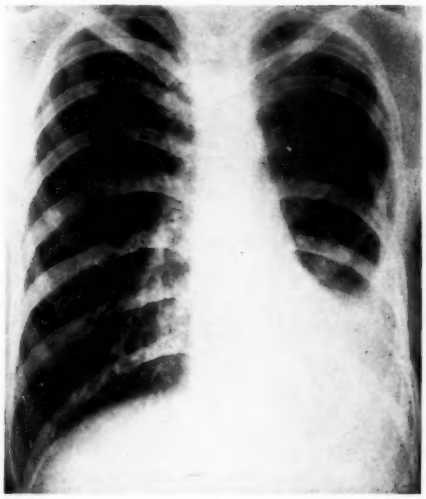


Fig. 9. Case 2. Immediate final result. The basal opacity is due to residuals of the small complicating empyema which required drainage.

entire lung surface, including the line of the lingular resection. At the points where this coat came near the stumps of the hilar elements, however, such intimate dissection seemed more hazardous than was justified, even though, no doubt, complete freeing of the lung base in this area would have been advantageous. Nevertheless, this decortication permitted the lung to expand very adequately on increasing intratracheal pressure, which was not at all possible before. The chest was closed and the pleura was again drained by an anterior and posterior catheter. The postoperative result was very gratifying (Fig. 8), there being prompt expansion of the lobe and progressive filling of the hemithorax by lung. A small basal empyema did develop, requiring drainage through the posterior catheter opening.

The patient was discharged with the empyema healed and with only minimal basal residuals visible by roentgenogram (Fig. 9).

Neither of these cases would permit the diagnosis of massive organizing hemothorax or fibrinothorax as the usual traumatic case was envisioned (9). In Case 1, the pleural content was aspirable readily and easily, the air replacement being necessary for control of negative pressure symptoms only. Case 2 did reveal a residual intrapleural mass by x-ray examination, in addition to the shadow cast in the roentgenogram by the collapsed upper lobe, but again the bulk of pleural content was aspirable in satisfactory fashion.

Nevertheless, these cases illustrate the points in diagnosis required to establish the fact that the lung could not expand because of a retaining pleural factor.

1. There was no evidence of bronchial obstruction.

2. There was no known intrapulmonary factor in Case 2 and no reasonable probability of such in Case 1.

3. The demonstrable pleural factor in Case 1 could be neutralized by aspiration and at least partially neutralized in Case 2 by means of the same method. No evidence of bronchopleural fistula could be adduced in either case.

4. Both patients had a pleural factor reasonably capable of producing a retaining peripleural coat—blood in Case 1 and serofibrinous pleural fluid in Case 2.

SUMMARY

1. The factors governing lung re-expansion after collapse are reviewed and a working classification of them is given.

2. The essential points in establishing the diagnosis of a lung that is retained in the collapsed state by pathological peripleural investments are discussed, and the relation of this process to organizing hemothorax or fi-

brinothorax as seen in thoracic wounds of violence is shown.

3. Two cases are reported in which these principles in diagnosis and their effective treatment by thoracotomy and decortication are illustrated.

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A NEW ESCHAR TECHNIQUE FOR LOCAL TREATMENT OF BURNS

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THE principal objectives in the treatment of burns have been well summarized by Allen and Koch and by Green, and it has been pointed out by them that the primary aim is to save life, and to restore the function and the appearance of the injured individual to as near normal as possible.

Since this article will deal only with the local therapy of the burned area, all of the objectives in the treatment of thermal injuries will not be listed but only those which are directly related to the wound. These objectives are listed below and they should be attained, when feasible, by the most efficient, the simplest, and least complicated method:

1. The prevention or minimizing of shock—a reduction in the plasma volume during the period of shock is prevented by minimizing the internal and external loss of fluid, salts and protein.
2. The relief of pain, both the immediate pain and subsequent pain due to dressings.
3. The prevention or minimizing of infection (early and late).
4. The use of a local form of therapy which will not interfere with the patient's recovery either because of undesirable systemic or local effects.
5. The reduction of the external and internal protein and salt loss to a minimum during the convalescent period.
6. The induction of healing as promptly as possible or in the case of a third degree burn the preparation of the burned area for grafting at the earliest possible date.
7. The provision of rest for the injured part.
8. The provision of a form of treatment in which the progress of the wound can be expeditiously observed without fear of contamination.

During the past 10 years, two methods of treatment have been widely used, namely that of applying a chemical coagulant which forms an eschar (3, 5, 14) and the method employing petrolatum or an ointment on the burned surface with an overlying pressure dressing (4).

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In a recent article, Bettman (6) presented his results on the treatment of over 700 patients with 5 per cent tannic acid and 10 per cent silver nitrate. In his series, the mortality rate was 3.1 per cent and he pointed out that several patients with very severe burns survived (over 70 per cent of the body surface involved). The cause of death in this series could in no instance be attributed to the toxic properties of tannic acid. In a group of patients which the authors regarded as being too small to permit any definite conclusions, Saltonstall, Walker, Rhoads, and Lee had a lower mortality rate in those treated by the tannic acid method than with pressure dressings. However, they also showed by liver function tests that the tannic acid, and other chemical eschar methods, produced some impairment of liver function which was not evident in the patients treated by the vaseline pressure dressing method. Experimentally, these findings have also been corroborated by Clark and Rossiter.

Rae and Wilkinson pointed out that the extent and likelihood of liver damage is much greater when tannic acid jelly is employed than when the tannic acid-silver nitrate method is used. The report by McClure, Lam, and Romence substantiates this view. The latter group as well as Allen and Koch, showed a significant decrease in the mortality rate when the tannic acid method was abandoned for the nonadherent pressure dressings.

It is not the purpose of this paper to discuss the relative merits of these two forms of therapy, but to point out that they are not entirely adequate, at least by a theoretical analysis, and to present a new form of treatment which more completely fulfills the objectives of the local burn therapy here outlined.

METHODS AND RESULTS

On the theory that animal tissue might provide a suitable and effective substance which

would form a more physiological eschar than tannic acid or the other tanning agents do, numerous extracts of various tissues were tried. An extract made from beef aorta reacted in the desired way. The protein thus obtained can be used in saline or in an ointment containing glycerine and diglycol stearate (it can also be made with 5 per cent sulfathiazole, or by adding one hundred thousand units of penicillin to 20 to 30 grams of ointment). The purified extract of the beef aorta is a protein, but is apparently free of albumin, proteoses and peptones. This extract in the treatment of burns has been under experimental and clinical study for several years and has been used in various vehicles (saline solution or paste containing several different antiseptics).[1] I have employed this treatment in over 500 ambulatory cases, and it has also been used in an additional group of over 250 patients with more serious burns by Weller and Zyla.

Judging from these clinical observations and studies on animals, the protein extract from the beef aorta reacts in the following manner:

a. It forms a dry slightly flexible eschar over denuded surfaces of the skin and if necessary can be softened by water or saline solution. This property permits it to be removed very easily without damage to the underlying tissue. Other workers (1, 20, 31, 32) have described similar findings in employing plasma or fractions of plasma for the local treatment of burned areas.

b. The extract in saline solution, or made up in the form of a salve containing sulfathiazole, sodium 2-hydroxydiphenyl, glycerol and diglycol stearate, does not injure tissue (7, 10) and shows no evidence of retarding tissue growth. Figure 1 shows a histopathological section taken 6 hours after an application of the protein aortic-extract in an ointment containing 5 per cent sulfathiazole following the removal of the epidermis from the skin of a pig by a dermatone. This section showed very little reaction in comparison to like sections which were taken after the employment of tannic acid or a vaseline pressure dressing (7, 9).

c. This material can be applied locally by spray or paste or incorporated into fine mesh gauze and a dressing employed (with or without pressure). Since the eschar separates readily when infection or epithelization occurs, its removal presents no problem.

d. The protein extract with or without added antiseptics, adheres to the burned skin surface and remains dry until the wound has healed or the slough begins to liquefy. The same observations have been made by others using a similar method (1, 31).

e. Weeping surfaces show a prompt decrease in the loss of fluid after application of the protein extract, which also occurs when other types of eschars are used (18, 26, 32).

f. It rapidly relieves pain when applied to first, second, or third degree burns.

g. If the bandaging method is employed, one application is sufficient, and at any time within a few hours, inspection of the area may be carried out by raising the outer protective dressing. No discomfort is experienced and the lesion is protected from air-borne infection because of the eschar. Infected areas can be easily recognized as the eschar liquefies and disintegrates (similar properties have been noted (1, 10, 20, 31, 32) when fractions of human plasma have been used).

h. Gauze impregnated with the ointment will form an eschar which is slightly flexible and capable of withstanding considerable trauma and pressure.

The eschar formed on denuded surfaces with this form of treatment approaches in appearance that which normally occurs when the skin is injured mechanically (32).

If the eschar is broken, tissue fluid exudes which promptly closes the opening by forming a scab, thus sealing off the wound.

In third degree burns, it has been noted that the slough separates at least as early as when other forms of therapy are employed. Since early inspection of the wound is possible, without pain or fear of contamination, skin grafting can be accomplished at the earliest possible date. The extract will not cause any irrita-

[1] This extract is prepared by grinding the beef aorta and then washing it to remove the excess blood. The washed material is then extracted with one-tenth normal sodium hydroxide solution for one hour and filtered. The filtrate is reduced to a *p*H of approximately 4.2 by the addition of dilute hydrochloric acid and the precipitate thus formed is then recovered by filtration. This product then may be redissolved in a physiologic solution of sodium chloride and used as a spray, or it can be worked into a paste or ointment.

tion nor will it firmly adhere to the normal epidermis, hence the outline of the injury is depicted by the adherent protein. When the eschar desquamates from a second degree burned area, the tissue beneath is soft. In a deeper burn as the necrotic tissue separates, the underlying granulations are clean, firm and red. (Edema of the denuded area is absent or minimum unless infection occurs.) Small areas of infection which may be present in fairly extensive burns can usually be recognized quite early. They can be treated by sponging the area with saline or any water soluble antiseptic and after it has been well cleaned the region can be again covered with the protein extract. The properties of this extract have been tested to determine if it produces any allergic reactions. (The details of these tests will be published later. However, no undesirable results have been encountered clinically or experimentally.)

The aorta extract in the form of an ointment, or as a solution should be applied to the burned area preferably after it has been thoroughly cleansed.

If the blisters are broken but the wound is clean, the injured skin can be pulled over the denuded area. Small unbroken blisters are left intact and large blisters are drained. A method of cleansing the wound by spraying using a standard seltzer bottle to which is attached a rubber tube carrying a DeVilbiss spray head has been used satisfactorily. The cleansing solution which has been found satisfactory is an antiseptic detergent diluting one part of the detergent to ten parts of sterile water. One carbon dioxide capsule may suffice but two capsules will completely empty the bottle at a more satisfactory pressure. The *p*H of the antiseptic detergent is on the acid side, and works well with the aortic extract. Following the use of the antiseptic detergent the wound is flushed with sterile normal saline. If this is not done the eschar usually is too thick. If the eschar becomes too firm this can be relieved by applying an ointment with an oily base. The ointment or solution containing the extract is then applied and the patient is placed in a heat tent, or a box splint may be employed for burns of the extremities in ambulatory patients. When dressings are used, the ointment can be applied directly to the burned area which is then covered with fine mesh gauze, or it can be incorporated in the gauze which is placed over the burned area. Several layers of dry gauze are then applied and bandaged.

Other observations concerning the therapeutic possibilities of this protein extract are of interest. When it is applied to the early vesicles of herpes simplex, it adheres to the small vesicles which usually desquamate in about 24 hours, leaving a smooth but erythematous area, which will appear entirely normal after the extract is reapplied and desquamation again occurs. An erythema resulting from sunlight or ultraviolet light will be relieved and the pain alleviated if the protein extract is applied early. The flare and accompanying discomfort resulting from many types of insect bites is also relieved in a few minutes following the application of this material, while on the other hand, the vasodilation resulting from histamine hydrochloride shows no response to the application of the protein extract. The aortic tissue extract does not delay or accelerate the coagulation of whole blood. However, it does delay coagulation of thoracic lymph.

DISCUSSION

In order that the method just described can be evaluated from a theoretical standpoint, it is best compared to other forms of treatment in relation to the objectives outlined.

1. The prevention or minimizing of shock—reducing the external and internal fluid loss. Experimental studies have shown that the eschar method (18, 26, 32) will prevent the external loss of fluids, electrolytes, and protein and that the nonadherent pressure dressing (8, 34, 36) will reduce the formation of edema in the injured area, if applied early. However, local edema without external loss may occur when tanning agents are used, and a plasma-like fluid may seep from the injured surface for days when pressure dressings are employed. Since an eschar which does not injure tissue can be produced and at the same time a pressure dressing may be employed, the protein extract should be the preferable method.

While pressure dressings may be used, cotton or mechanic's waste have been found to interfere with the formation of an adequate eschar. Because many of the patients exhibit some edema prior to the time the wound is dressed, and since pressure dressings are only effective on the extremities, the open method, or the closed method employing loose dressings, are usually used.

2. The relief of pain—immediate and subsequent pain. Following local treatment with all three methods, pain is usually readily relieved, but since the removal of a tannic acid eschar or changes of a pressure dressing are often uncomfortable, the aorta extract is preferable.

3. The prevention or minimizing of infection—early and late. All three methods if carefully employed should be quite adequate. During the first few weeks, however, when dressings are changed the danger of introducing air-borne bacteria should be less when an eschar is present. While there is some controversy over the desirability of applying the sulfonamides or antibiotics locally, many workers (2, 25, 27) feel that such therapy is of some benefit. On the other hand, Meleney failed to demonstrate a statistically significant reduction in the incidence of infection in a large group of patients that had local applications of a sulfonamide, when compared to a control group of similar cases. However, he does state that it seems likely that the use of the sulfonamides may have minimized the spread of the local infection, and therefore reduced the incidence of septicemia and death. Whether bacteria can be inhibited or not by such means is not within the scope of this discussion. It should suffice to say that a bacteriostatic substance can be employed with a nonadherent pressure dressing or with the protein eschar method, if desired.

The following points should be kept in mind if the sulfonamides are to be employed: (a) They should not be used to replace careful surgical technique (15). (b) They should not be dusted on an open wound in a powder or crystalline form or used in a water dispersible base (15, 16, 24), but should be employed only where a limited, slow absorption of the drug will occur. A 5 per cent suspension of

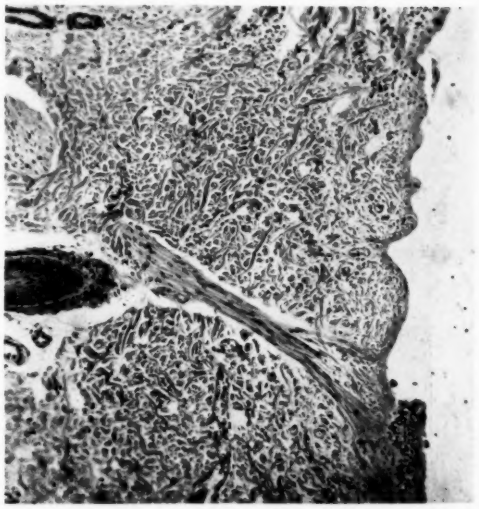

Fig. 1. A histopathological section taken 6 hours after application of the protein aortic extract. A uniform thickness of epidermis was removed from a young pig. Sections were taken from a control animal and when various other forms of treatment were employed. This section shows little reaction in comparison to like sections treated by other methods (7, 28).

sulfathiazole in aortic tissue ointment applied to 13 per cent of the body surface on rabbits subjected to second degree burns and the surface mechanically denuded, showed an average blood level in 6 animals of 1 milligram of free sulfathiazole per 100 cubic centimeters of blood in 2 hours; the same in 6 hours; and a trace in 7½ hours. The highest level encountered at any time was under 2 milligrams. Levels indicating that the absorption was too rapid were seen when sodium sulfathiazole was used, so this was not employed (10).

Since the pressure dressing method has not yet been sufficiently evaluated, disadvantages may still become apparent. In Bettman's (6) cases, infection occurred more frequently when a greasy ointment was applied locally. Experimentally (19) the incidence of Curling's ulcer was found to occur more often in animals treated by the vaseline pressure dressing method (66 per cent), than when tannic acid or other eschar forming substances were employed (6 per cent). However, the pressure dressing method is not suited for animal experiments, and infection seemed to be the

Fig. 2. Case J57. Age 1¼ years. Subjected to second degree burns of the back, buttocks, posterior thighs, lower half of abdomen and upper right arm, on January 1, 1943, when he fell in a bucket of hot water. Child was treated for shock with saline and plasma 5 hours after the accident. Burned area was douched with saline. The aortic tissue extract ointment was applied to the abdomen with a layer of gauze. Patient was placed in a heat tent, face downward, and the tissue extract ointment was applied to the back, buttocks, thighs, and right arm. Eschar formed in 10 hours. Eschar began falling off on the 11th day and all areas were completely healed on the 14th day. (Courtesy of Dr. C. N. Weller.)

major factor in causing the high incidence of Curling's ulcer.

4. The use of a local therapy which has no undesirable, systemic or local effects. The nonadherent dressing or the protein eschar method has no known undesirable systemic or local effects. It has been demonstrated conclusively that tannic acid and other tanning agents will cause some impairment of liver function (35). It has also been pointed out (37) that if a patient is placed in a tub containing tannic acid, or if the jelly is employed (29), the danger of liver necrosis is a real hazard. Clinical studies (9, 21) and experimental animal work (28) have shown that healing is impaired and some normal tissue destroyed when tannic acid or other chemical coagulants are used.

Fig. 3. Case J60. Suffered second degree burns of face, neck, scalp, and ear, January 4, 1943. Wounded area was douched with normal saline 1½ hours after being burned with hot gravy. The aortic tissue extract ointment was applied. The eschar was formed in 12 hours. Tissue extract was applied to the chest for the second time on January 5, 1943. Lesion was completely healed January 13, 1943. (Courtesy of Dr. Weller.)

5. The reduction of the external and internal protein loss. It has been shown (12, 22) that the amount of protein or nitrogen lost externally from a wound may be quite large, and Cuthbertson has again recently emphasized the importance of this loss. For the reasons enumerated in the first part of the discussion, the protein eschar method is preferable. Even when the open method is used, the edema does not seem to be as great as that observed with other methods. Some studies have been made with the use of this type of

therapy and although not complete they seem to indicate, contrary to what other observers (12, 22) have found when pressure dressings are employed, that the external nitrogen loss is negligible from the area of a second degree burn.

6. The induction of healing as promptly as possible and the preparation of the third degree areas for grafting at the earliest possible date.

From a study of a limited number of cases, it appears that the healing and grafting processes are possibly accelerated when the protein eschar method is employed. The experimental and clinical studies by Hoffman and Dingwall and the observations of others (31, 32) help corroborate this impression. Therefore, it is believed that this method can be considered at least as efficient as the nonadherent pressure dressing method.

7. The providing of rest for the injured part. This could be accomplished by any of the three methods of therapy, but it should be easier by the protein eschar with dressings or splints, or with the nonadherent pressure dressing method.

8. The providing of a form of treatment in which the progress of the wound can be expeditiously observed without fear of contamination.

Experience with the protein extract method indicated that this form of local therapy is superior to the two commonly employed methods. When the vaseline pressure dressings have been used, patients with fever and without wound infection have frequently had the dressings removed unnecessarily for examination of the burned area. On other occasions, with the pressure dressing or tannic acid eschar methods, the infection has spread rapidly and extensively before the wound was observed.

If the protein eschar method is employed, the open treatment can be used, or if dressings are applied, they can be easily removed and the burned area can be inspected frequently. If a small area under the eschar becomes infected, it softens or liquefies, thus indicating the presence of infection. It can then be cared for early and further spread prevented.

SUMMARY

1. A new method for the local treatment of burns has been described.

2. This method has all of the advantages offered by the other available methods and no known disadvantages.

3. It has been effectively employed in over 500 ambulatory burned patients. Since these patients were not extensively burned, no definite conclusions can be drawn regarding the results of this treatment on the mortality rate, but there is reason to believe that it should be at least as effective as the other available forms of local therapy.

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PULMONARY EMBOLISM BY AMNIOTIC FLUID

Report of 3 Cases with a New Diagnostic Procedure

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IN 1941 Steiner and Lushbaugh (5) described maternal pulmonary embolism by amniotic fluid, thereby adding another entity to the pathology of parturition. DeLee maintained that, "The number of possibilities this discovery suggests is uncountable at present" and he called it "an exquisitely interesting discovery." In most of the cases thus far reported, this catastrophe occurs during the course of labor or the immediate puerperium and is characterized by the appearance of sudden profound shock followed by the death of the mother and frequently of the fetus. Postmortem examination reveals no significant gross finding to explain the cause of death. However, the microscopic appearance of the lungs is diagnostic. The sections exhibit a relatively bloodless vascular bed in which many of the vessels are occluded by polymorphonuclear leucocytes and the particulate constituents of amniotic fluid (epithelial squamae, mucin, granular débris, etc.). A variable degree of pulmonary edema may be associated with this condition.

A total of 10 cases was reported by Steiner and Lushbaugh, 8 in 1941 (5) and 2 in 1942 (3). No other report of this disease could be found in the literature. Very recently 1 additional case was described by Burt.[1]

In this communication we hope to rekindle interest in this condition with the presentation of 3 new cases and a procedure whereby the diagnosis can either be verified at autopsy or perhaps established when autopsy is impossible.

CASE REPORTS

CASE 1. A 25 year old white woman, secundipara, was admitted to the obstetrical service (Dr. J. W. Stevenson) in active labor 21 days after the expected date of confinement. She had had one pregnancy 8 years previously which was normal. The past medical history was not significant. The present pregnancy had been normal. Eleven hours previous to admission, bloody vaginal discharge appeared followed in 2 hours by the first labor pains 15 minutes apart. About 9 hours later the membranes ruptured spontaneously. On admission uterine contractions were tetanoid in character, occurring at 5 minute intervals and lasting 20 seconds. Rectal examination revealed 3.5 centimeters' dilatation of the cervix with moderate effacement. The fetus was in the left occipitoanterior position of a vertex presentation and had a heart rate of 130 per minute. No significant abnormalities were found on routine physical examination. The blood pressure was 118/76. Temperature, pulse, and respiratory rate were within normal limits. The results of urinalysis and serologic tests were negative.

One hour after admission the patient was given demerol, 100 milligrams and scopolamine, grains 1/150 (0.4 mgm.) hypodermically. One hour and 45 minutes later the cervix was fully dilated and effaced. There was an emesis of undigested food particles at this time. Immediately after being placed upon the delivery table the patient became restless and complained of "chilliness." The skin was cold and clammy. This was followed by severe hyperpnea and coma. Within 15 minutes the patient was apneic pulseless, and intensely cyanotic. There was conjugate deviation of the eyes to the right. The blood pressure was 30/00. Emergency measures evoked no response and death occurred 40 minutes after the onset of symptoms. A dead, cyanotic fetus was delivered during the last 5 minutes by version and extraction.

The autopsy, performed 2 hours after death, yielded only nugatory gross findings. However, the blood within the heart and vessels was everywhere fluid. No clots were observed. The left and right lungs weighed 225 and 270 grams respectively. There was a small amount of frothy, blood-tinged mucus in the left bronchial tree. The uterus was normal for its puerperal state. The placenta was not available for examination. The other organs were grossly normal.

Microscopically, the arteries and most of the arterioles of the lungs are occluded by a mixture of polymorphonuclear leucocytes, monocytes, acidophilic granular débris, and basophilic fibrillar material, and contain few or no red blood cells. The leucocytes are frequently arranged in parallel rows as though raked or combed. The endothelial lining of the vessels is normal and no leucocytes are found within the vessel wall or in the perivascular tissue. Epithelial squamae (Fig. 1) and occasional bits of

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[1]While this paper was in press another case report appeared. Hemmings, C. T. Am. J. Obst., 1947, 53: 303-306.

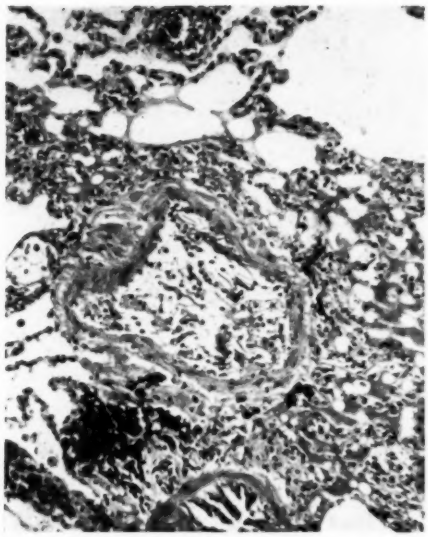

Fig. 1. A bloodless pulmonary artery from Case 1 which contains epithelial squamae, mucus, and leucocytes. Hematoxylin and eosin stain. × 150.

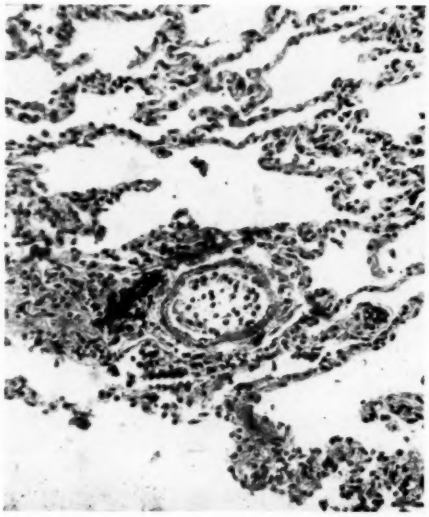

Fig. 2. A smaller pulmonary arterial branch from Case 2 which contains leucocytes and granular material but no blood. Hematoxylin and eosin stain. × 150.

bile-stained granular débris are present in a few small vessels. The alveolar spaces are empty except for a few which contain a small amount of fluid. The bronchi are normal.

The liver sections show slight separation of the sinusoidal endothelium from the liver cords by albuminous fluid. Sections of the spleen exhibit severe passive hyperemia. Sections of other organs, including the uterus, show no significant abnormality.

CASE 2. A 42 year old white woman, tertipara, was admitted to the obstetrical service (Dr. J. C. Hughes) 10 days before the expected date of confinement. There were 2 normal pregnancies 20 and 22 years previously. The past medical history was essentially negative.

At 6 months of the present pregnancy, the patient was hospitalized because of pernicious vomiting and intermittent uterine contractions. At this time she was poorly nourished, moderately dehydrated, and had lost considerable weight. Physical examination disclosed no abnormality other than a mild dermatitis over the anterior abdominal wall. The results of urinalysis and of serologic tests were negative. A moderate secondary anemia was present. After 6 weeks of supportive therapy and bed rest she was discharged much improved and had no recurrence of vomiting.

The patient was readmitted at term with rhythmical contractions every 20 minutes and some bloody vaginal discharge. The fetus was in a vertex presentation with the head at the pelvic inlet. The cervix was thick and 2 centimeters dilated by rectal examination. Fetal heart sounds were of good quality in the left lower quadrant. The membranes were unruptured. The uterus appeared excessively enlarged and its contractions were strong. Physical examination revealed no significant abnormality. The blood pressure was 120/70; pulse, temperature, and respiratory rate were normal. Urinalysis yielded no abnormal finding.

Because of the excessive uterine distention, an abdominal binder was applied, the membranes were ruptured artificially and about 1 liter of amniotic fluid was drained off. During the following 4 hours little progress was made and 6 pitocin injections were given, 1 minum and 2 minum doses, alternating every half hour. This was ineffective and labor advanced slowly during the next 24 hours despite a repetition of the pitocin injections. Thirty-four hours after admission she began to have severe tetanoid contractions and made rapid progress. Two hours later the cervix was fully dilated and effaced. Fetal heart sounds were of good quality at this time. One hour later, the patient was delivered of a stillborn male infant by outlet forceps under nitrous oxide-oxygen-ether anesthesia. Immediately following delivery the patient became cyanotic, hyperpneic, and exhibited evidence of severe shock. The placenta was delivered without difficulty. The patient became progressively more cyanotic, clammy, and pulseless and died 65 minutes after the onset of symptoms despite immediate antishock treatment.

Autopsy was performed 2½ hours after death. On opening the abdomen, about 200 cubic centimeters of

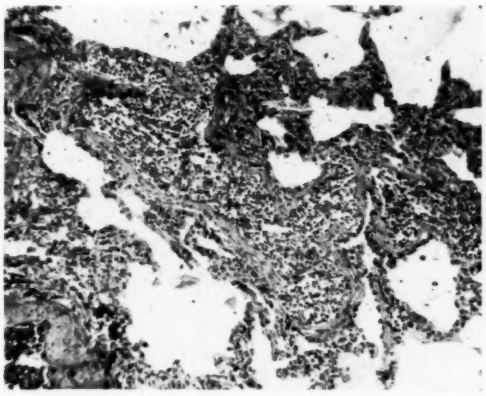

Fig. 3. A pulmonary artery from Case 3 which is plugged by masses of leucocytes and granular material. Several epithelial squamae and some mucus are also present. Hematoxylin and eosin stain. × 90.

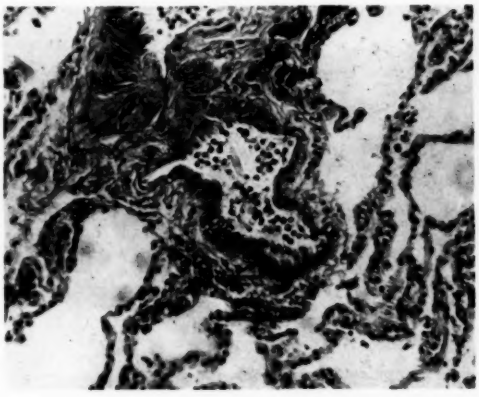

Fig. 4. A pulmonary artery from Case 3 which contains a hair shaft (lanugo) in addition to leucocytes as well as a small amount of débris. Hematoxylin and eosin stain. × 140.

bloody fluid were encountered. There was diffuse bloody extravasation into the preperitoneal tissues of the anterior abdominal wall below the umbilicus. There was also ecchymosis at the root of the mesentery and the mesenteric attachment of the terminal ileum and appendix, extending laterally to the cecum. This ecchymosis also involved the right broad ligament of the uterus with the corresponding tube and ovary. All these tissues were edematous. The blood within the heart and vessels was everywhere fluid; no clots were observed.[1] The left and right lungs weighed 175 and 210 grams respectively and were grossly normal. The liver was normal except for severe pallor. There was moderate dilatation of the left ureter and the left renal pelvis. The uterus was normal for its puerperal state. The placenta was not available for examination. Other organs were normal.

Microscopically most of the pulmonary arterioles and alveolar capillaries are bloodless, and are occluded by polymorphonuclear leucocytes and a small amount of granular débris (Fig. 2). Some of the larger vessels are similarly involved. Mucus as seen in Case 1, is demonstrable in a medium-sized artery, but no squamae are seen. The endothelial lining of the affected vessels is intact and the cells are not swollen. There is no indication of leucocytic migration within or outside of the vessel walls.

The liver cells are swollen and the sinusoids narrowed. The latter contain only occasional red blood cells and present instead, many polymorphonuclear leucocytes and monocytes. An occasional small portal vein shows similar cellular content together with a small amount of acidophilic, granular débris.

No significant abnormality is noted microscopically in the other organs.

Case 3. A 35 year old white female, quintipara, was admitted to the obstetrical service (Dr. J. J. Weber) in active labor 1 month prior to the expected date of confinement. There had been 4 previous full term pregnancies 15, 10, 5 (stillborn), and 3 years previously. The past medical history was essentially negative. The present pregnancy was uneventful and the patient entered labor in an apparently healthy state.

The first labor pains began about 2 hours before admission, occurred at 10 minute intervals, and were accompanied by a slight bloody show. On admission the contractions occurred at 5 minute intervals and lasted 30 seconds. Fetal heart sounds were of good quality at 140 per minute. The fetus was in left occipitoanterior position of a vertex presentation. The head was unengaged at the pelvic inlet. The cervix was very soft and dilated 3.5 centimeters. Physical examination revealed no significant abnormalities. The results of urinalysis were normal. The blood pressure was 130/80. Temperature, pulse rate, and respiratory rate were normal.

The patient was given demerol, 100 milligrams, and labor proceeded uneventfully for 6 hours. At the end of this time the cervix was fully dilated and effaced and the head was at the level of the ischial spines. The membranes ruptured spontaneously. It was noted at this time that the amniotic fluid contained fresh blood. Fifteen minutes later the delivery of a normal, live infant occurred spontaneously. The third stage lasted 4 minutes and was uneventful. Pituitrin, 1 cubic centimeter, was given after the second stage and ergotrate, grains 1/320, (0.2 mgm.) after delivery of the placenta. No anes-

[1]Blood removed from the right auricle was incoagulable. It was placed in a refrigerator but unfortunately, was not examined until 2 weeks later. After centrifugation, the sediment contained, in addition to the usual red cell layer and leucocytic cream, a top stratum of white flocculent material. Although shreds suggestive of mucus were found in smears made of this top layer, identification was not positive and the results were considered equivocal.

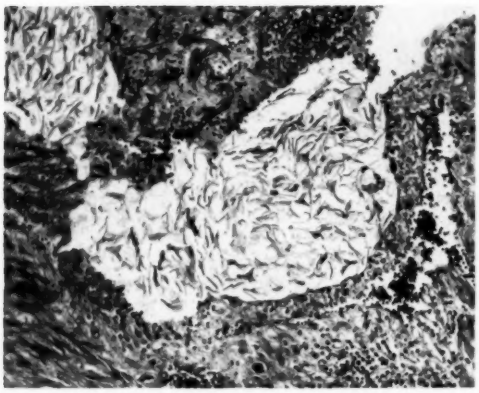

Fig. 5. Case 3. In the uterus was found a large venous sinus filled with great masses of epithelial squamae. Hematoxylin and eosin stain. × 90.

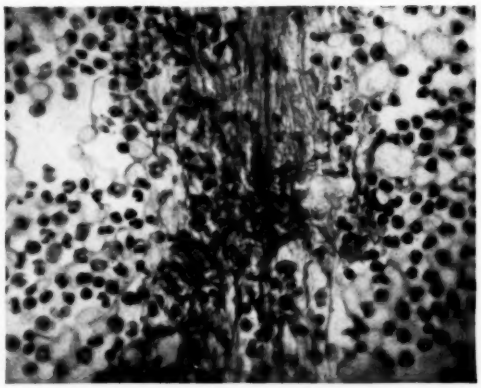

Fig. 6. A smear of the top, flocculent layer of blood sediment from the inferior vena cava of Case 3. A band of mucus characterized by its transparent, veil-like nature shows fine, long, parallel fibrils. Wright's stain. × 355.

thetic was administered. For 20 minutes following delivery, the patient was alert and communicative. Then there was an emesis of green-tinted fluid. This was followed 5 minutes later by stupor, severe hyperpnea, and coldness and clamminess of the skin. The blood pressure was 40/20. The patient became comatose and pulseless within 15 minutes and respirations were slow and irregular.

At this time about 500 cubic centimeters of bloody fluid were expressed from the uterus and it was believed that shock had occurred from uterine hemorrhage. However, blood removed by venepuncture 25 minutes before death had a hemoglobin content of 10.8 grams per 100 cubic centimeters. The plasma protein content was 5.8 grams per 100 cubic centimeters and the hematocrit was 32 by the copper sulfate specific gravity method. Death occurred 1 hour after the onset of symptoms despite antishock treatment.

Autopsy was performed about 1 hour after death. Immediately following the primary incisions and exposure of the abdominal and pleural cavities blood samples were aspirated from the inferior vena cava and from the pulmonary artery. A clean, sterile syringe with a No. 18 gauge needle and clean, sterile specimen bottles with rubber stoppers were used for this purpose.

After autopsy, it was felt that death could not be explained on the basis of the gross findings. The uterus was normal for its puerperal state. The placenta and membranes showed no abnormality. There was extensive thrombosis of veins of the broad ligaments. These thrombi were soft, of currant jelly type, and not attached to the vessel wall. Elsewhere the blood was thin and fluid and no clots were found. The heart weighed 285 grams. Aside from moderate dilatation of the right auricle no abnormality was noted. The lungs were dry and spongy, weighing 150 and 180 grams respectively. They were partially collapsed and entirely normal externally and on section. Other organs were essentially normal.

Microscopically there are extensive vascular occlusions beginning with the medium-sized pulmonary arteries and extending down to the alveolar capillaries. The lumina of these vessels are filled with a mixture of loosely packed leucocytes and acidophilic granular material (Fig. 3). Longitudinally-cut hair shafts (lanugo) are found in two of the larger arteries (Fig. 4). This hair is demonstrable in 7 consecutive serial sections of one of these vessels. In 2 smaller arteries the leucocytes appear to be "combed-out" in parallel rows or lines containing in addition, fine acidophilic granules and a few epithelial squamae. This "combed-out" appearance of the leucocytes and granules is characteristic of mucus within the vessel. No fat is demonstrable in frozen sections stained with sudan IV.

Although the above findings are present throughout both lungs, they are not uniformly distributed. Nevertheless, there is very little blood in the pulmonary vascular tree. There is no pulmonary edema. The alveolar spaces and bronchi are empty, but in some sections slight atelectasis is present.

In the uterus a very significant lesion is found. A large blood sinus near the endometrial surface contains large masses of epithelial squamae (Fig. 5).

The liver is noteworthy microscopically because of detachment of sinusoidal endothelium from the liver cords by albuminous fluid with narrowing of the sinusoidal spaces and general ischemia.

Sections of thrombotic veins from the broad ligament show no abnormality of the vessel wall. The thrombi are of very recent origin, show no organization, and no attachment to the endothelium.

No significant abnormality is found in sections of the other organs.

Blood studies. The blood aspirated at the beginning of the autopsy did not coagulate. After centrifugation, done within 3 hours after death, the sediment showed 3 distinct zones instead of the normal 2. There was a broad, more flocculent zone of lighter color above the thin gray layer of leucocytic cream.

Smears were made of the top layer of sedimented material after the supernatant fluid had been removed. These smears were air-dried and stained with Wright's stain and with mucicarmine. The latter stain requires alcohol-fixation. The flocculent top layer was then transferred to a small tube and compressed by centrifugation. A portion was fixed in alcohol and the remainder, in formaldehyde, and each was embedded in paraffin. The fixed middle, or leucocytic, layer was similarly embedded. Sections were cut and stained with hematoxylin and eosin. Those from the alcohol-fixed block were also stained with mucicarmine and phosphotungstic acid-hematoxylin.

Both smears and sections show the bulk of the upper flocculent layer to be composed of finely granular acidophilic material containing widely scattered small clusters of leucocytes. The smears also show occasional, single, long chains or rows, of 10 to 20 leucocytes. In one smear of the top, flocculent layer of blood from the inferior vena cava, an occasional elongated mass of mucus is found (Fig. 6). The mucus is characterized by its transparent, veil-like nature showing very fine, long, parallel fibrils all of which stain pink with mucicarmine. There are many eosinophiles in and about the masses of mucus.

In the sections of the alcohol-fixed blood sediment, which were stained with hematoxylin and eosin there are small clumps of fine and coarse granules of golden yellow (bile!) color. Thin, wavy fragments, characteristic of squamae, are found in the sections but not in the smears. These stain red with eosin and yellow-brown with phosphotungstic acid-hematoxylin. (It is to be remembered that the latter stains fibrin blue.)

Smears and sections of the middle zone which consists of leucocytic cream, show no abnormal findings.

DISCUSSION

The work of Steiner and Lushbaugh (5) has established maternal pulmonary embolism by amniotic fluid as a distinct pathological entity which is to be suspected clinically in every case of sudden, severe shock during labor or the immediate puerperium. These authors pointed out that this catastrophe is one of the most common causes of death "during labor and within the first nine hours thereafter." This opinion was based on an incidence of 1 in 8,000 obstetric cases over a period of 9 years at the Chicago Lying-in Hospital. However, because the diagnosis was made only in cases in which autopsies were performed the frequency of occurrence is probably considerably greater. It is an almost incredible, but a significant coincidence that the 3 cases reported here occurred consecutively within 1 year in a general hospital averaging 1200 to 1300 deliveries annually, and they represent *all* the maternal deaths on the obstetrical service within that time.

Steiner and Lushbaugh (5) contended that many unrecognized nonfatal cases do occur. Furthermore, in patients on whom autopsies were not done it is probable that a sizeable percentage of maternal deaths labeled thrombotic pulmonary embolism, obstetric shock, or postpartum hemorrhage are actually fatalities due to pulmonary embolism by amniotic fluid. Even after a postmortem examination it is possible that the disease can escape recognition unless it is kept in mind.

At the present time, the clinical diagnosis cannot be made before the appearance of shock. In the cases here reported the patients entered labor with no apparent complication except for moderate hydramnios in Case 2. In Cases 1 and 2 tetanoid contractions were observed late in the second stage. It was noted by Steiner and Lushbaugh (5) that such contractions were a frequent prelude to the shock state. In Case 3 the amniotic fluid contained blood. This supports the theory that the immediate predisposing factor of this disease is a communication between the uterine venous sinuses and the amniotic cavity. Shock occurred suddenly and without warning near delivery or soon thereafter, and was irreversible. In 2 of these cases death was attributed clinically to postpartum hemorrhage since much bloody fluid was expressed from the uterus after shock was manifest. However, the amount of hemorrhage was small, and the hematocrit, hemoglobin content, and the plasma protein value of the antemortem blood sample in Case 3 were not consistent with exsanguination. The phenomenon of blood incoagulability described in terminal shock (4) was a striking feature in all 3 cases.

The gross findings of the postmortem examination of these 3 patients and of all the cases thus far reported have failed to explain the cause of death. Microscopically the disease is limited to the pulmonary arteries, arterioles, and capillaries. The involved vessels are

bloodless and are occluded by emboli consisting of abundant polymorphonuclear leucocytes, mucin, bile-stained débris (meconium), epithelial squamae, lanugo hair, and granular débris with or without fatty elements. The character of the embolic picture is variable. Which of these materials are present in any given vessel depends upon the proportion of meconium, vernix caseosa, and débris in the amniotic fluid and upon the heterogeneous distribution of these elements in the circulating blood. Often enough the vascular obstructions in the smaller branches consist of leucocytes alone. The source of these leucocytes is obscure. Inasmuch as they are also found in great numbers in the larger pulmonary arteries their origin is probably extrapulmonary.

The sections of liver from Cases 2 and 3 show the microscopic picture of serous hepatitis (Roessle) which occurs frequently in cases of sudden death following shock. The liver sinusoids and portal venules of Case 2 contain polymorphonuclear leucocytes and granular material, a picture similar to that seen in the lung.

Blood, from cases of pulmonary embolism by amniotic fluid, removed from the right side of the heart or the inferior vena cava, and subjected to centrifugation may show 3 strata in the sediment. The presence of 3 instead of 2 strata should be considered pathognomonic of this condition. The particulate constituents of amniotic fluid including mucus, being of low specific gravity, settle out as a flocculent layer above the leucocytic cream. It is recommended that both smears and sections be made of this layer. The sections should be cut from alcohol, as well as Zenker, or formalin-fixed blocks of this sediment. The stains should include Mayer's mucicarmine for the identification of mucus, Mallory's phosphotungstic acid-hematoxylin for the differentiation of fibrin from mucus and epithelial squamae, and hematoxylin and eosin for routine purposes.

The utility of this procedure is twofold. In cases in which permission for autopsy cannot be obtained, it may be possible to secure blood from the right heart by aspiration. This blood examined by the method described may become the only means for a positive diagnosis. In cases in which autopsy permission is granted this procedure serves to re-enforce the diagnosis.

Such postmortem aspiration and examination of blood from the right heart, adopted as a routine procedure in obstetrical deaths, may furnish desirable information regarding the frequency of embolism by amniotic fluid.

SUMMARY AND CONCLUSIONS

Three cases of pulmonary embolism by amniotic fluid are reported. They were characterized by the sudden, unexpected development of irreversible shock during labor or the immediate puerperium, and were followed very quickly by death. At autopsy, no significant gross abnormality could be found. Microscopically, the diagnosis was established by the demonstration of extensive vascular occlusions by leucocytes and constituents of amniotic fluid in the smaller branches of the pulmonary arteries.

The sediment of blood aspirated from the pulmonary artery and the inferior vena cava of one of these cases showed a pathognomonic, third, flocculent stratum above the layer of leucocytic cream. In smears and sections of this stratum, constituents of meconium and amniotic fluid are demonstrable.

In cases of obstetrical death, routine aspiration and examination of blood from the right heart by the method described, may establish the diagnosis of pulmonary embolism by amniotic fluid when autopsy cannot be performed. In this way the frequency of this condition may be determined more accurately.

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THE PLASTIC SURGICAL CLOSURE OF DECUBITUS ULCERS IN PATIENTS WITH PARAPLEGIA

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New York, New York

THE excision and closure of decubiti in patients with paraplegia by plastic surgical methods is one of the interesting and valuable contributions made by military surgeons during World War II. The first report of bold surgical attack upon an anesthetic bedsore in the paralytic was published by Lamon and Alexander in 1945. However, it is stated in the literature that the case cited was housed in the neurosurgical section of the McCaw General Hospital which was under the direction of W. B. Scoville. Many who are familiar with the work of Scoville at the Cushing General Hospital during the recent war feel that priority should be given him for introducing the idea of the surgical treatment of decubitus. Undoubtedly Charcot's concept (1879) of a neurogenic, trophic factor presumed to be responsible for the development of decubitus in denervated areas caused surgeons to hesitate to attack these lesions surgically in civilian practice as well as in military practice in the early years of the war.

As these unfortunate wounded accumulated in Army hospitals during the war, Scoville's work became known. Subsequent reports have attested to the value of the methods of plastic surgical excision of decubiti in paraplegics. In 1945, White, Hudson, and Kennard (15) reported 5 such cases in which patients were treated successfully by excision and the advancement or rotation of flaps of adjacent skin and subcutaneous tissue. Croce, Schullinger, and Shearer (1946) reported 8 cases in which the decubiti were excised and the defects were closed by the advancement of multiple regional flaps of skin and subcutaneous tissue. Poer (1946) reported on the incidence of decubiti in patients paralyzed due to war injuries. Forty-four (57.1%) of 77 patients with gunshot injuries of the spinal cord had decubiti. Barker, Elkins, and Poer (1946) reported their results in 70 cases in which decubiti were treated by simple excision and suture, by rotation flaps, or by the free grafting of skin. White and Hamm (14) (1946) published their experiences with 14 decubiti in 9 patients treated by simple excision and closure or by sliding flaps. Gibbon and Freeman reported on the operative closure of 65 ulcers in 43 patients.

Much information has been gained from a study of these reported groups of cases. The organized plan of the United States Army Medical Department for the care of paralyzed individuals, as outlined in Poer's publication, has resulted in obtaining the maximum benefit of neurosurgery for these patients, and a high percentage of them have been spared the serious complications of the genitourinary tract. They have been benefited by exercise, by physical therapy, by general supportive measures, and some have been completely rehabilitated. Many of these patients have now been transferred to Veteran's hospitals where they continue to receive the benefits of physical therapy and are undergoing rehabilitation. The closure of long persistent or recurrent decubiti in these chronic cases is the present problem of paramount importance. The continued loss of protein and nitrogen from open wounds is the causative factor underlying the debility of the patients. The excellent studies of Mulholland, CoTui, Wright, Vinci, and Shafiroff demonstrated that, in patients with decubiti, the plasma protein concentrations invariably are below normal and that a negative nitrogen balance exists.

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Read before the Surgical Section of the New York Academy of Medicine, February 7, 1947.

Because the surgical treatment of decubiti in paraplegics involves the transposition of soft tissues supplemented occasionally by the use of free grafts of skin, this surgery has been the responsibility of the plastic surgeons. At the Veteran's Administration Hospital in the city of New York there have been 65 patients with paraplegia, 59 of whom have had 298 decubiti. This report deals with the surgical management of these patients. Their ulcers have been of long duration, resistant to conservative treatment and, in a few cases, recurrent after surgical excision and repair. In comparison with the reports referred to above it is apparent at once that the decubiti in this group of patients are of longer duration than those encountered in Army and Navy hospitals during the war. Included in this group of patients are those who have failed to be rehabilitated physically and mentally, some who have been lacking in morale and in cooperation, and a few who have developed harmful habits such as alcoholism. Despite these circumstances the results following surgery have been gratifying.

ETIOLOGY

There is general agreement that pressure over osseous prominences such as the trochanter, the sacrum, or the anterior superior spine is the most important single factor in the development of decubitus. Compression of the soft tissue overlying bone causes local anemia which, if sufficiently prolonged, results in necrosis. Also there is general agreement that old age, general debility, avitaminosis, malnutrition, and anemia are contributing factors.

However, there is difference of opinion regarding the pathogenesis of decubitus in paraplegia. Gibbon and Freeman, basing their conclusions on the work of Brown-Sequard, Paget, and Cohnheim feel that neurogenic factors are not responsible but that decubiti in paraplegics are due to continued local pressure associated with loss of motion and of muscle tone. Munro, elaborating on the neurogenic theory first advanced by Charcot, feels that the development of a decubitus in a paraplegic is dependent on the effect of abnormal vascular reflexes caused by disturbance of the sympathetic nerves of the area. He points out that in his series of cases with injury to the thoracic and lumbar spinal cord the incidence of bedsores was 54 per cent while in the cases with injury to the cervical cord the incidence was only 20 per cent. He states that there is failure of the proper response of circulation of the skin to its varying needs and that this failure is due to a reflex of the sympathetic nerves. According to this theory spinal shock, present immediately after injury, affects the peripheral vasomotor reflexes with the failure of development of increased regional flow of blood which is necessary for proper nutrition of the part. However, Munro agrees that all bedsores start as pressure sores and may be prevented by turning the patient hourly, by regular massage, by keeping the skin dry, and by the use of powder. MacCollum feels that pressure causes a local diminution of blood supply resulting in necrosis of the skin and underlying tissues. Boyd calls the condition a "pressure gangrene" which is influenced by a devitalized condition of the tissues as a result of old age, long illness, or of hypostasis secondary to feeble circulation. In spinal cord lesions, he believes that capillary stasis from injury to vasomotor nerves is a contributing factor.

PATHOLOGY

In the early stages of decubitus, continued pressure produces thrombosis of small blood vessels. In the affected area a reddish patch appears which soon changes to a dusky color and necrosis follows. Slough separates gradually to leave a granulating surface. The granulation tissue of the ulcer is infected and is grey in color. As the ulcer increases in size the slough extends farther in subcutaneous areas than in the adjacent skin so that one may be deceived concerning the size of the lesion. Also, the ulcer extends toward the deeper layers of tissue so that eventually the underlying bone is exposed. The appearance of the base and the undermined sides of an ulcer of long duration gives the impression of an open sac with a smooth lining. Photograph of a specimen of decubitus ulcer which was excised at operation is shown in Figure 1. The specimen has the gross appearance of a cyst

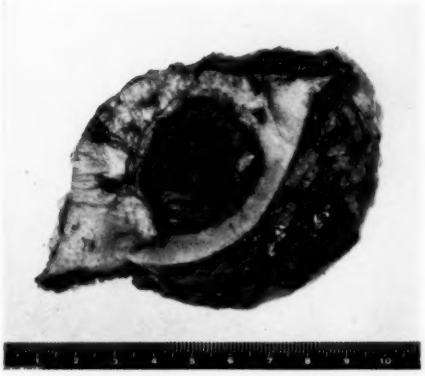

Fig. 1. Specimen excised from a patient with decubitus over the trochanter of the femur. Note the cyst-like character of the lesion.

with a relatively small opening through the skin. Its thick walls consist of fibrous tissue and its lining is of smooth, grey, avascular, granulation tissue. On microscopic section it is observed that the smooth appearance is due to a lining of necrotic tissue and pus superimposed on an area denuded of skin. The underlying and adjacent connective tissue is edematous but well vascularized. There is infiltration of polymorphonuclear leucocytes, lymphocytes, and plasma cells with occasional histiocytes. The arterioles have thick walls. There is proliferation of fibrous tissue throughout. The capillaries and venules are dilated and engorged. Undoubtedly, this stasis of blood contributes to the extension of the ulcer. Photomicrographs of ulcer tissue are shown in Figures 2 and 3.

BACTERIOLOGY

The causative organisms assume an important rôle when the appearance of the ulcer indicates spreading infection and when there is evidence of systemic reaction. The necrotizing skin and underlying tissues are contaminated with bacterial flora of the skin and intestinal tract. These invade the deeper tissues as the process extends. The presence of anaerobic streptococci may cause an inflammation which progresses rapidly with resultant extensive gangrene. Careful anaerobic and aerobic cultures should be taken in such

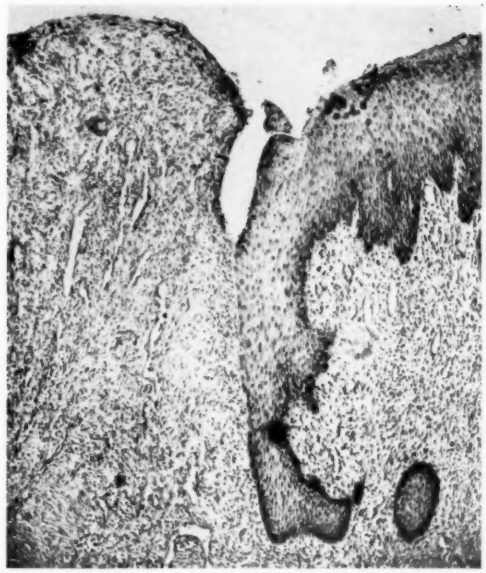

Fig. 2. Low power photomicrograph of the edge of a chronic decubitus. The marginal epithelium is embedded in dense fibrous tissue.

cases so that specific treatment may be instituted immediately after excision of the gangrenous tissue. Also it is advisable to deter-

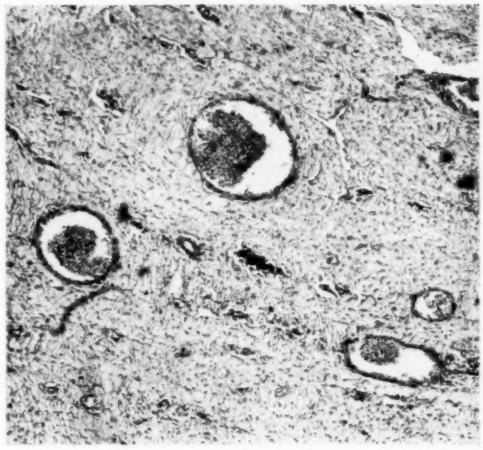

Fig. 3. High power photomicrograph of tissue from the floor of a decubitus of 12 months' duration in a patient with paraplegia due to gunshot wound of the spinal cord. Marked capillary dilatation and stasis is in evidence.

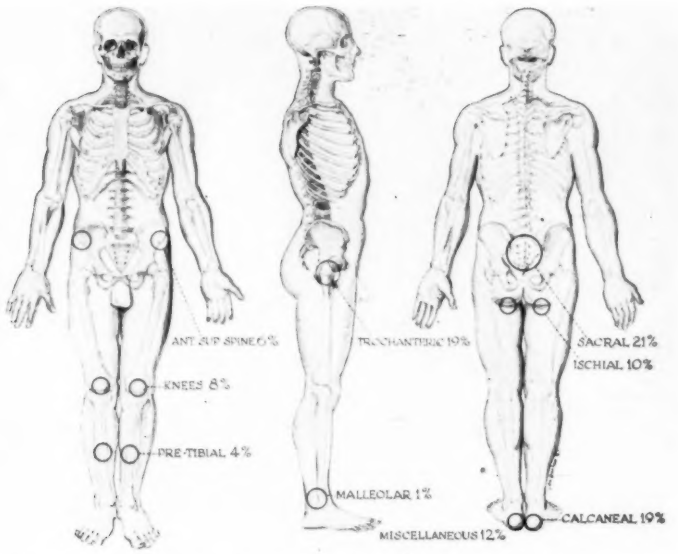

Fig. 4. Diagram showing the locations of 298 decubiti in 50 patients with paraplegia due to war injuries.

mine the susceptibility of the causative organisms to penicillin, to streptomycin, and to the new antibiotic, bacitracin.

In the indolent ulcers one finds a wide variety of organisms. Anaerobic and aerobic studies were made of the flora of the ulcers in 18 cases in this series. A variety of organisms were reported in the following order of frequency: staphylococci (usually coagulase positive), Bacillus proteus, Escherichia coli, Bacillus pyocyaneus, diphtheroids, hemolytic streptococci and nonhemolytic streptococci. In addition, in 9 of the cases the anerobic plates showed the pinpoint colonies of microaerophylic streptococci. Bacteriologic studies made by Gibbon and Freeman and by White and Hamm (14) revealed similar findings. In the cleansing of an indolent decubitus ulcer in preparation for surgery the frequent change of saline compresses has been found to be most useful.

Specific types of infection have called for specific local therapy. Dressings of zinc peroxide have been very effective in clearing up areas where there was gangrene of tissue in association with anaerobic organisms. Application of parachlorophenol has been used effectively to combat heavy growth of the Bacillus pyocyaneus. Nine-amino acridine hydrochloride is effective against most of the other organisms which have been recovered from decubiti in this series of cases.

PREOPERATIVE AND POSTOPERATIVE CARE

In preparation for surgery a high caloric diet with extra nourishment is given in an attempt to restore a positive nitrogen balance. Transfusions of blood, of plasma, and protein digests are indicated for the treatment of hypoproteinemia and anemia. Vitamins (B complex and C), liver extract, and iron are administered. Patients with good appetite and steady gain in weight are considered to be ready for operation. Only those patients in whom complications of the urinary tract have been avoided or controlled are subjected to operation.

Many of the patients in this series demonstrated involuntary spasms of skeletal muscles. In several such cases these spasms constituted a factor which contributed to the development of complications in the wounds

Fig. 5. a, above, Small decubitus over the anterior superior spine of the ilium in a patient with transection of the spinal cord at the level of twelfth Thoracic. b, Result after elliptic excision of the decubitus and linear closure of the defect. This method was found useful only in the management of small ulcers. It was successful in 74 per cent of the cases in which it was used.

following surgical closure. Such spasms have resulted in partial or complete separation of the edge of a wound and in a few of these cases bursae have developed under the flaps of soft tissue. Therapy with penicillin and sulfonamides is given for 24 hours prior to operation. The preparation of the wound includes daily cleansing with saline sponges and the application of a fine mesh gauze impregnated with xeroform ointment. Slough is excised at the time of each dressing. Specific infectious agents require the use of specific local therapy as mentioned above. Cleansing enemas are given prior to operation.

In the care after operation the measures described in the preparation for operation are continued. Penicillin is administered throughout the postoperative period. The position of

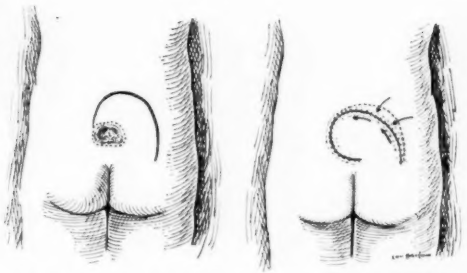

Fig. 6. Diagram of the method of excision of decubitus ulcer and closure by pedicled flap.

the patient when returned to bed must be supervised carefully. The success of surgery depends upon the avoidance of those factors which in themselves contributed to the development of the original decubitus. The patient must be moved from side to side and from prone to supine. It is inadvisable to allow the weight of the body to cause pressure over the operative site until the wound is inspected at the time of the first dressing. Gross soiling of the operative area is prevented by the use of a retention catheter, by administration of a low residue diet, and by the giving of liberal doses of paregoric. Enemas are withheld until the 7th postoperative day. The first postoperative dressing is done on the 6th, 7th, or 8th day after operation at which time alternate sutures are removed. The last sutures are not removed until the 14th day after operation. Collections of fluid, or clot are aspirated and solution of penicillin is instilled.

STATISTICS

Sixty-five patients with paraplegia are included in this analysis. Fifty-nine of these have had 298 decubiti. Six patients had no pressure sores. Of these 59 patients all but 3 had unhealed decubiti at the time of admission to this hospital. Forty-nine had multiple decubiti while only 10 patients had a single decubitus. Six of these patients had lesions of the cervical cord; 53 had lesions of the dorsal or lumbar spinal cord. Of those with lesions of the cervical cord 3 exhibited multiple ulcers, 1 patient had only 1 ulcer and 2 patients had none at all.

Of interest is the location of the 298 decubiti which occurred in the 59 patients (Figure 4).

Fig. 7. a, above, Appearance of chronic decubitus over the sacral region in a patient with paraplegia due to transection of the dorsal spinal cord. b, Result after excision of the decubitus and closure by rotation of a pedicled flap with primary closure of the defect created by the construction of the flap. Objection to this method is based on the fact that tension at the line of suture laterally may be reflected to the medial line of suture which resides close to the site of the original decubitus. This method was successful in 62.5 per cent of cases in which it was used.

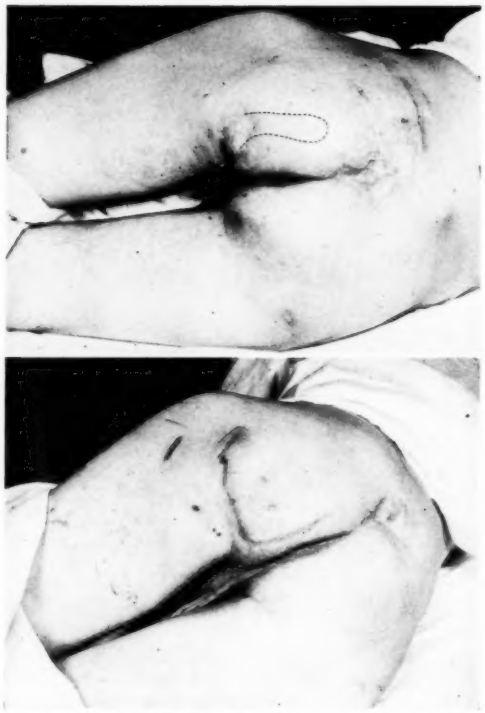

Fig. 8. a, above, Decubitus of left buttock in patient with paraplegia due to gunshot wound of dorsal spinal cord. The relatively small external opening with deep extension into the underlying tissue was a common characteristic of the decubiti over the ischial tuberosities in the group of cases included in this report. b, Appearance after excision of the ulcer and sinus and closure by advancement of a flap from neighboring tissue as shown in Figure 6. This method was successful in only 62.5 per cent of the cases in which it was used.

The most frequently encountered decubitus was that of the sacral region. Sixty-three (21 per cent) were in this area. There were 57 (19 per cent) ulcers over the trochanters. Also, there were 57 (19 per cent) over the os calcis. It is of interest that 26 patients had ulcers over both heels while only 5 showed ulceration over only one heel. With one exception these ulcers responded to conservative therapy, healing spontaneously. There were 30 ulcers (10 per cent) over the ischial tuberosities, 23 ulcers (8 per cent) over the knee, 19 ulcers (6 per cent) over the anterior superior spine of the ileum, 11 ulcers (4 per cent) in the pretibial areas, and 38 ulcers (13 per cent) were distributed in various areas of the paralyzed torso or extremities.

TABLE I.—STATUS OF 298 DECUBITI IN 59 PATIENTS

	No. ulcers	Per cent
Healed by conservative treatment	189	63.4
Unhealed at time of this analysis	53	17.7
Healed following excision and closure	40	13.4
Persistent following excision and closure	12	4.0
Recurrent following excision and closure	4	1.3
Total	298	

An analysis of those cases in which the ulcers were treated by excision and plastic surgical closure is of interest. Thus far, 56 ulcers over the sacrum, the trochanter, the ischial tuber-

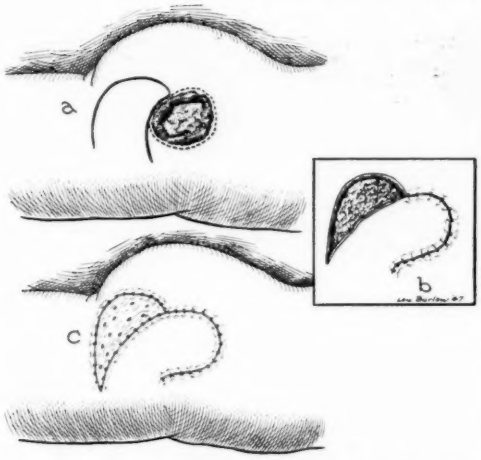

Fig. 9. Diagram of the method of excision of decubitus and closure by pedicled flap with free thick-split graft at a distance. The method has the advantage that lines of suture are without tension and do not reside over the area of primary decubitus. The free graft effectively provides a permanent surface for the soft tissues underlying it. This method was successful in 75 per cent of the cases in which it was used.

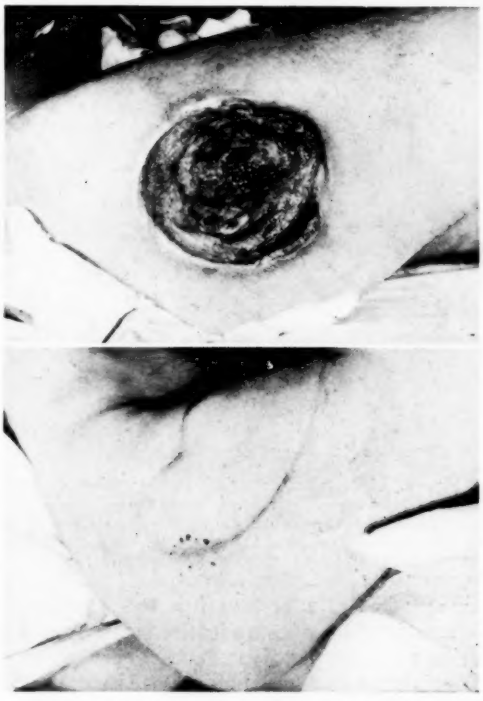

Fig. 10. a, above, Decubitus of 10 months' duration over the trochanter of a patient with paraplegia due to gunshot wound of the spine at the level of the 4th thoracic segment. Fragmented bone was exposed in the base of the ulcer. b, Result after excision of decubitus and closure by pedicled flap with free thick-split graft at the site of elevation of the flap. Note that by the use of this method the graft resides permanently over soft tissues, not over a bony prominence.

osity, the tibia, and the anterior superior spine have been operated upon (Table I). The largest ulcer encountered in this series of cases was 12 by 15 centimeters. In a few cases the ulceration was quite small but often such an ulcer represented the opening of a deep sinus. In one case such a sinus with an opening only 1 centimeter in diameter was found to extend to a depth of 14 centimeters. Forty, or 71 per cent, are healed following surgery. In these patients the ulcers had been present for an average time of 12 months. In 16, or 29 per cent, the ulcer persisted or recurred following excision and closure. In these patients the average length of time between the onset of the ulcer and the date of operation was 15 months.

OPERATIVE TREATMENT

The operative treatment of decubitus consists of the complete excision of the ulcer and adjacent scar and the covering of the defect with an adequate pad of healthy tissue. By this is meant a covering of skin with a thick layer of subcutaneous fat. Usually this can be advanced from surrounding tissues but conceivably might be transplanted from a distance.

The complete excision of the ulcer may be a difficult technical procedure. Ulcers of long duration are surrounded by a dense layer of scar and usually the ulceration has undermined the regional soft tissues extensively. Subcutaneous excavation of the ulcer may extend as far as 14 centimeters beyond the margins of the ulcer itself. Ulcers over the trochanter or ischial tuberosity often are associated with fragmentation and necrosis of bone necessitating partial ostectomy as a step in excision of the ulcer. Because of the profuse bleeding from scar tissue at the time of excision it has been our practice to administer a

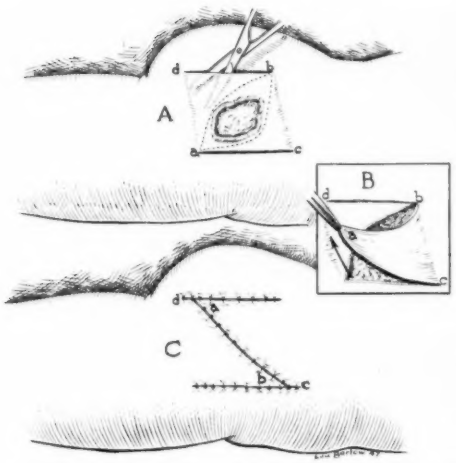

Fig. 11. Diagram of the technique of excision of decubitus and closure by Z-plasty. Because the defects following excision of decubiti usually are quite broad this method of closure is not considered to be ideal. It was successful in only half of the wounds to which it was applied.

transfusion of blood during the operation. Hematoma as a complication may lead to failure of the operative effort. Therefore emphasis must be placed on accurate hemostasis. Electrocoagulation has been used for this purpose but the authors feel that its use is not ideal since it leaves traumatized or necrotic tissue in the wound. Its use in this series of cases was followed by excessive accumulation of serum in the wounds. Hemostasis is effected best by the use of mosquito clamps and fine ties of No. 00000 plain catgut or No. 120 cotton.

The operative procedures which have been used in the 56 operations in this series are 5 in number. These procedures are based upon established principles of plastic surgery. They are as follows: (a) elliptic excision and closure; (b) excision and closure by Z-plasty; (c) excision and closure by pedicled flap; (d) excision and closure by pedicled flap with free graft at a distance; (e) closure by free skin graft. Emphasis has been placed on the necessity for covering the site of ulceration with a generous flap of tissue and without scars of suture overlying the original site of ulceration. To accomplish this a free thick-split graft of skin at the site of procurement of the flap has

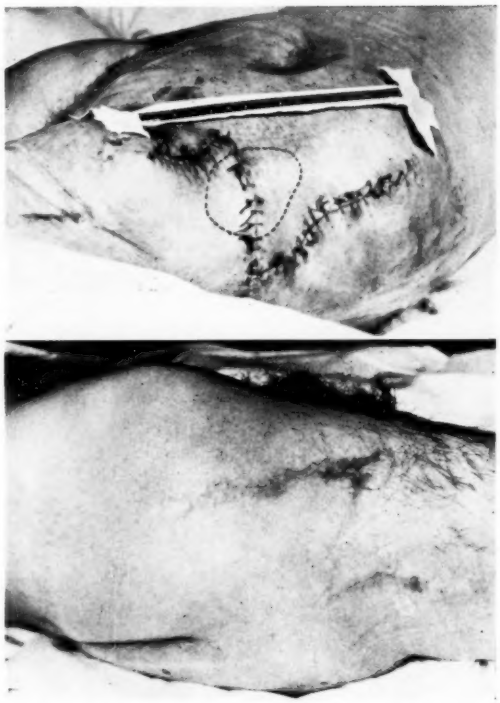

Fig. 12. a, above, The dotted circle represents the size of the decubitus over the trochanter in this case. The lesion was present for 12 months. It was excised and the defect closed by Z-plasty. b, Appearance 3 months after excision of the decubitus and closure by Z-plasty.

allowed for its rotation or advancement without tension. Free grafts applied directly to the decubiti have been used only in those cases in which the patient was too sick to undergo major surgery. Others have pointed out the value of this method calling attention to the facts that it provides wound closure and permits stabilization of altered body chemistry. Diagrams of steps involved in these five operative procedures are outlined in Figures 6, 9, 11, 13, and 15. Photographs of lesions before and after operation are shown in Figures 5, 7, 8, 10, 12, 14, and 16.

RESULTS

Elliptic excision has been used only in the management of very small ulcers. This method was applied in 23 cases with complete

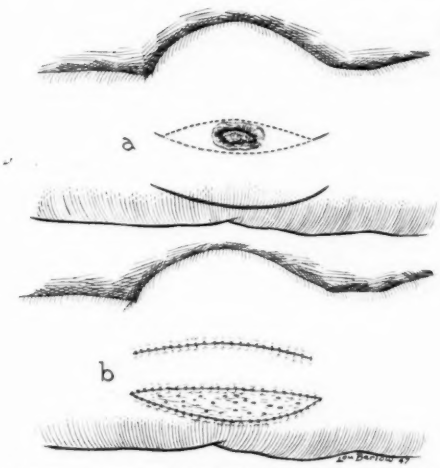

Fig. 13. Diagram of the method of excision of decubitus and closure by a double pedicled flap with free graft at a distance. This method represents a variation of that shown in Figure 9 and it has the same advantages.

Fig. 14. a, above, Appearance of decubitus of left trochanteric region in a patient with paraplegia due to a lesion at the level of the eleventh thoracic segment of the cord. b, Appearance 6 weeks after excision of the decubitus and closure by the advancement of a double pedicled flap with free graft over the soft tissues from which the flap was raised as outlined in Figure 13.

healing in 17, or 74 per cent. The value of closure by Z-plasty has been limited. This principle does not often apply to closure following the excision of decubiti, as the resultant defect may be quite broad. Z-plasty was used in 4 cases. Three were trochanteric ulcers and 1 was an ulcer over the ischial tuberosity. The operation was successful in 2 cases and in 2 cases failure resulted.

Excision and closure with a pedicled flap and primary closure of the defect caused by rotation of the flap was successful in only 5 (62.5 per cent) out of 8 cases. Healing was effected by this operative technique in 2 cases of sacral ulcer and 3 cases of trochanteric ulcer. The failures occurred following the use of this method in 3 cases of ulcer over the ischial tuberosity. It is felt that the use of a free graft to cover the defect from which the flap was rotated might have effected satisfactory healing in these cases. Considering the size and location of the ulcers the impression has been obtained that excision and closure by a pedicled flap with a free graft at a distance is the best method available. By this method the thick-split graft resides permanently over soft tissue which is not subjected to the pressure of a bony prominence. This method was used in 16 cases with complete healing in 12 (75 per cent) and failure in 4 cases (25 per cent). It was used in the management of 6 sacral ulcers, 9 trochanteric ulcers, and 1 tibial ulcer. The use of free skin grafts (thick-split) has been reserved for those cases in which the patient was too sick to undergo major surgery. The value of the method lies in the fact that, with a minor operative procedure, a closed wound may be obtained with resultant stabilization of altered physiology. Though this method of closure is followed by improvement in the general condition of the patient, it does not provide a closure of sufficient sturdiness to withstand the constant trauma to which the area is subjected in the paralyzed patient. Therefore, it is looked upon as a closure which is of temporary value. It is used with the idea of excising the graft at a later date and covering the formerly ulcerated area with a thick

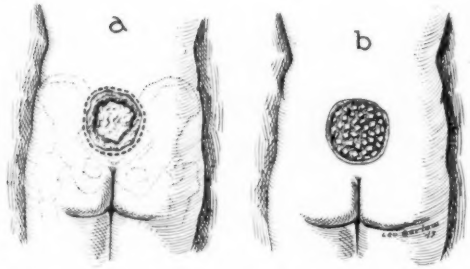

Fig. 15. Diagram of the method of temporary closure of decubitus by the application of multiple small thick-split grafts of skin. Free grafts were used only when the general condition of the patient was such that major operative procedure was contraindicated. Upon the degree of vascularity of the granulation tissue of the decubitus rested the decision as to whether the wound was surfaced by a single large graft or by multiple small grafts. The method allows for the early restoration of altered protein metabolism and therefore is useful as a temporary measure. Grafts were successful in 4 out of 5 decubiti to which they were applied. In recognition of the fact that free grafts cannot be expected to withstand daily trauma to bony prominences in paralyzed patients it is planned to excise such grafted areas at a later date and to cover the defect by a pedicled flap as diagrammed in Figure 9.

flap of skin and subcutaneous tissue. The fact that healing followed in only 4 of the 5 cases in which free grafts were used is not considered of statistical significance for the reason that the method was used only in the management of ulcers in those patients with extreme general debility.

Study of Table III shows that ulcers in the sacral area have been healed effectively by operative surgery in 88 per cent of cases. The closure of ulcers in the trochanteric area was successful in 72.7 per cent of cases. Ulcers in the region of the ischial tuberosity were closed successfully in only 46.6 per cent of cases in the series.

An analysis of the *complications* which resulted in failure in 16 cases following operative closure is of interest. These complications were as follows: hematoma of wound, 4 cases; undue tension in line of suture, 6 cases; uncontrollable spasm of extremities, 2 cases; massive infection, 2 cases; partial necrosis of flaps, 1 case; lack of co-operation of patient, 1 case.

DISCUSSION

Attention has been called to the fact that complete healing followed in 40 (71 per cent)

Fig. 16. a, above, Decubitus over the sacral area in a patient with paraplegia due to a lesion of the spinal cord at the level of the second thoracic segment. Despite continued supportive therapy with transfusions of plasma and blood augmented by the giving of amino acids and protein digests this patient had severe anemia and marked hypoproteinemia. For this reason it was not deemed advisable to subject this patient to major operative procedure. b, Appearance of the sacral area after closure of the decubitus by the application of multiple, small thick-split grafts of skin. The general condition of the patient improved rapidly following healing of the wound, and at the time of this report he is a candidate for excision of the grafted area so that the bony prominence may be protected ultimately by a thick pad of soft tissue.

of the 56 decubiti treated by excision and plastic surgical closure. Only those ulcers which had failed to heal after conservative management for a minimum of 10 months were subjected to surgery. It has been observed repeatedly that decubiti of long duration are associated with pallor of the granulation tissue, excessive scar in the floor and at the margins of the ulcers, and with thinning of the skin and subcutaneous tissue adjacent to the ulcers. In the report of Croce, Schul-

TABLE II.—RESULTS FOLLOWING EXCISION AND PLASTIC CLOSURE

Type of closure	No. cases	Complete healing	Failures
Elliptic excision and closure	23	17	6
Excision and closure by Z-plasty	4	2	2
Excision and closure by pedicled flap	8	5	3
Excision and closure by pedicled flap with free graft at a distance	16	12	4
Closure by free skin graft (thick-split)	5	4	1
Totals	56 (100%)	40 (71%)	16 (29%)

TABLE III.—RESULTS FOLLOWING EXCISION AND PLASTIC CLOSURE

Location of decubitus	No. cases	Complete healing	Failures
Sacral	17	15	2
Trochanteric	22	16	6
Ischial	15	7	8
Anterior superior spine	1	1	0
Tibial	1	1	0
Totals	56 (100%)	40 (71%)	16 (29%)

linger and Shearer, the interval of time between onset of the ulcer and the date of surgical excision in 7 patients averaged 80 days. Barker, Elkins, and Poer reported complete healing following surgery in 39 (76.4 per cent) of 51 cases. The interval of time between the onset of the ulcer and date of operation is not given. Because their patients were treated in Army hospitals during the war it is presumed that the majority of the decubiti were of short duration. White and Hamm (14) reported complete healing following surgery in 6 (42.8 per cent) of 14 cases, partial healing in 6 (42.8 per cent) and failure in 2 cases (14.2 per cent). Gibbon and Freeman reported primary healing following surgery in 44 (67.7 per cent) of 65 cases, delayed healing in 10 cases (15.3 per cent), improvement in 5 cases (8 per cent), failure in 5 cases (8 per cent), and death in 1 case (1.5 per cent). In view of the fact that in the cases reported herein the ulcers were of long duration and had exhibited no tendency to heal under conservative measures, the success of surgery in 71 per cent of cases attests to the soundness of surgical attack upon chronic decubiti in patients who are suffering with paraplegia.

SUMMARY AND CONCLUSIONS

Report is made of experience with the excision and plastic surgical closure of 56 decubiti in paraplegic patients in a veteran's hospital. The locations of decubitus ulcers studied, their etiology, pathology, and bacteriology are discussed.

Five methods of operation have been used: elliptic excision and closure; excision and closure by Z-plasty; excision and closure by pedicled flap; excision and closure by pedicled flap with free graft at a distance; closure by free thick-split graft of skin. Forty, or 71 per cent, of the 56 cases were healed by these procedures.

All of the ulcers in this series were of long duration. They had been present for an average of 12 months in 40 cases in which plastic surgical closure was successful. The ulcers had averaged 15 months in duration in 16 cases (29 per cent) in which plastic surgical closure was not successful. In the management of this group of patients emphasis has been placed upon the application of the established principles of plastic surgery to the closure of the decubiti. Experience has indicated that methods in which the scar of suture resides directly over the site of the former decubitus are apt to be unsuccessful. Elliptic excision was used only in the management of small ulcers. Free graft of skin was used only in the care of ulcers in patients whose general condition would not permit major operative procedure. Evidence in this report indicates that large ulcers are closed successfully in the highest percentage of cases when a flap of skin and subcutaneous tissue is rotated from an adjacent area and the defect at the site of procurement of the flap is surfaced by a free graft of skin.

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SARCOMAS OF THE SMALL AND LARGE INTESTINE

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THE basis of this paper is a study of 15 cases of sarcoma of the small and large intestine: this includes 9 cases of sarcoma of the small intestine, 5 of the colon, and 1 of the rectum. It comprises all the cases collected from the records of the Jewish Hospital in the past 25 years. During the same period there were admitted to the hospital 48 patients with carcinoma of the small intestine, 1,377 with carcinoma of the colon, and 577 with carcinoma of the rectum. According to these figures the relative proportion of sarcoma to carcinoma of the small intestine is approximately 1 to 5.5, of the colon, 1 to 275, and of the rectum, 1 to 577. It appears, that even in the case of the small intestine, the relative incidence of carcinoma is considerably greater than that of sarcoma although it is generally believed that sarcoma of the small intestine is of more frequent occurrence than is carcinoma. The number of cases of sarcoma presented in this series may be too small to draw any definite conclusions as to the incidence of this lesion in the bowel, but it is nevertheless quite significant. Many of the observations recorded here are largely confirmatory of the information already available in the voluminous literature on this subject (3). However, additional emphasis will be placed upon those aspects which appear to warrant it.

There are no definite pathognomonic signs, symptoms, or laboratory criteria on which a diagnosis of sarcoma of the bowel can be made before operation. The onset of the disease, although usually insidious, may be acute, and the symptoms may vary in duration from several days to several years. These patients present various forms and locations of their tumors. A study of the cases reveals that the lesion may occur in all age groups, although it is most frequent in middle or past middle life. The youngest patient in our series was a 16 year old girl, and the oldest a 72 year old male, the average age incidence being 54 years. More than 65 per cent of the patients fell in the age group of 50 to 70 years. These figures are in close agreement with other figures reported in the literature.

It is generally conceded that sarcoma of the intestine occurs more frequently in males (1). In the cases reviewed here there was no appreciable difference in the distribution of the disease in the two sexes. There were 8 males and 7 females, showing a slight preponderance among males.

It has been the observation of many investigators that the incidence of the ratio of sarcoma of the small intestine to sarcoma of the large intestine is approximately 2:1 (2). In the series reported here, there were 9 instances of sarcoma of the small intestine, 2 of the appendix, and 3 of the large bowel, a proportion of approximately 2 to 1 in favor of the small intestine.

The etiology of sarcoma of the bowel is still unknown, although it is believed by some that trauma may play a part in its development. So far as the cases reported here are concerned, no history of injury was recorded in any of the patients.

A study of the cases reported in this series has emphasized in our minds the difficulties one encounters in being certain that a given tumor is sarcomatous or carcinomatous. The onset of the disease was usually insidious in character, and clinical evidence of its existence did not appear until late. When the onset was acute and the symptoms of short duration, there was always present some complicating factor to account for its acuity. Thus, in one patient with lymphosarcoma of the appendix, and in another with a neurofibrosarcoma of the rectum, the disease was heralded by a profuse intestinal hemorrhage associated with dizziness, fainting, and profound weakness. Two other patients with lymphosarcoma of the ileum developed acute intussusception and

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only then they first became aware of the existing disease. The majority of the patients, however, pursued a more chronic course which lasted from several weeks to 2 years.

Blood in the stools appearing as gross hemorrhage or melena, was a significant finding in 8 patients; in 2 others it was not reported. Patients with bleeding usually complained of weakness and dizziness, and on physical examination showed a variable degree of pallor which was proportionate to the loss of blood. The hemoglobin content of the blood was low and in one instance reached a level of 37 per cent requiring several blood transfusions. The nutrition was fairly well maintained in most of the patients, although some complained of a moderate loss of weight.

Pain, frequently described as cramp-like in character, was significant in 11 of the 15 patients recorded. The distribution of the pain varied, although it was usually confined to the area involved by the tumor. Anorexia, nausea and vomiting were infrequent complaints. Constipation, diarrhea, or both were more common. Many of the patients with constipation resorted to cathartics, and this may account for the occurrence of diarrhea in some of the patients.

A palpable mass was noted in 9 of the 15 patients. The location of the mass varied and was not always constant. In 4 instances it was felt in the right lower quadrant of the abdomen, in one, around the umbilicus, right upper, and left lower quadrants, respectively. In one instance the mass was palpated in the anterior wall of the rectum, and in another, it was visualized as a cauliflower, bleeding growth in the sigmoid, on sigmoidoscopic examination. In many cases the mass was tender to palpation, and was fixed in position or freely movable.

Roentgenography was of considerable aid in the diagnosis of these tumors. The roentgenologic appearance of the lesion showed great variations in different cases. In some it presented a mere filling defect or a moth eaten irregularity in the bowel wall. In others, the affected bowel appeared as a rigid tube or showed abnormal retention of barium.

In none of the cases reported here was a correct clinical diagnosis made before operation. Among the various diagnoses made preoperatively were colitis, carcinoma, ovarian cyst, bleeding duodenal ulcer and subacute appendicitis. It is significant that the tumor simulates a number of closely allied diseases of the intestine and renders correct diagnosis most difficult.

In this study, the following types of intestinal sarcoma were encountered: lymphosarcoma, 4; leiomyosarcoma, 7; reticulum cell sarcoma, 3; neurofibrosarcoma, 1; a total of 15. The tumors were found in the following locations: jejunum, 2; ileum, 7; appendix, 2; colon, 2; sigmoid, 1; and rectum, 1.

It is the consensus that lymphosarcoma is more frequent of occurrence than are the other forms of sarcoma of the intestine; this holds particularly true in the case of the small intestine. It did not hold true, however, in the present series. While the lymphosarcoma and reticulum cell sarcoma were particularly common in the small intestine, the leiomyosarcomas on the other hand, were equally common in the small and large intestine. No cases of lymphosarcoma were noted in the large bowel excepting in the appendix. Since lymphoid tissue forms an important part of the appendix, one should expect to find an appendiceal form of lymphosarcoma.

Leiomyosarcoma originates in the smooth muscle cells of the muscularis. It is usually slow growing and often well demarcated from the other layers of the intestinal wall. In the present series, there were 7 cases of leiomyosarcoma, 2 occurred in the ileum, 1 in the jejunum, 1 in the appendix, 1 in the cecum, 1 in the transverse colon, and 1 in the sigmoid. In 3 of the cases the lesion presented itself as a pedunculated polypoid growth, arising by a narrow or broad base from the inner surface of the bowel and partly encroaching upon its lumen. The free surface of the tumor was at times smooth and at others, necrotic and ulcerated. In the remaining cases, the neoplasm consisted of a homogeneous mass, grey or pink in color, and arising from the muscular coat of the bowel. The mucosa overlying the tumor was sometimes well preserved and apparently unaffected by the growth. On occasions, however, the mucosa was infiltrated with tumor tissue, became ulcerated, and

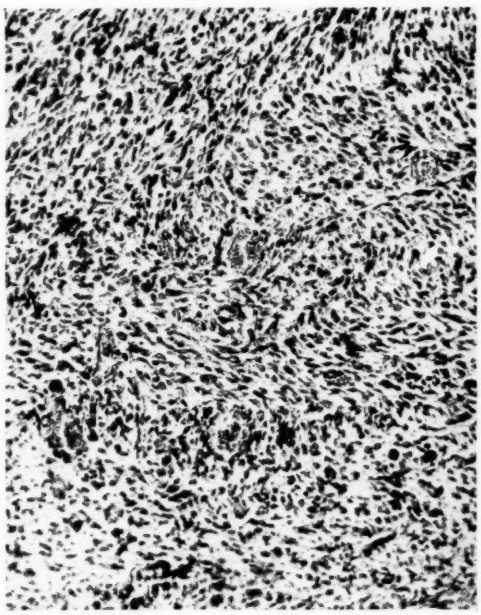

Fig. 1. Photomicrograph of leiomyosarcoma. Hematoxylin and eosin stain, ×80.

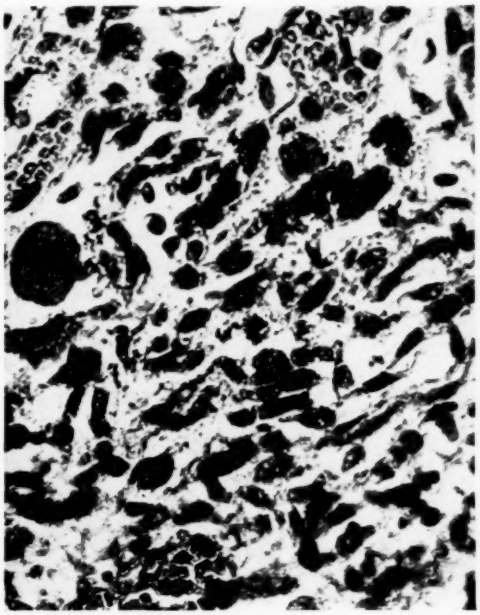

Fig. 2. Higher magnification of Figure 1. Note tumor giant cells. Hematoxylin and eosin stain, ×215.

showed scattered hemorrhages. The tumor was usually firm in consistency; in the more rapidly growing forms, it tended to become soft and necrotic. The direction of growth was not always uniform. In 2 cases it protruded into the lumen, and in the remaining ones it grew outwardly, forming a bulge on the exterior of the bowel.

Histologically, these tumors can be traced to the muscular coat of the intestine from which they are known to arise. Both in architectural arrangement and in cytologic detail, they almost regularly have features in common with the muscular layer and consequently they may readily be recognized as having arisen from this structure. In the case of the tumors presented in this paper, the parenchyma was made up of specific tumor cells which approached very closely the mother tissue. The cells formed interlacing bands or whorls running in different directions. They were spindle shaped or polygonal in outline, varying in size and shape and containing a round or vesicular nucleus. The nuclei were hyperchromatic and showed occasional mitoses. Scattered in the parenchyma were also found multinucleated giant cells. The stroma was very scant and consisted of a small amount of poorly vascularized, loose connective tissue (Figs. 1 and 2).

Although the tumor mimics very closely smooth muscle tissue, it may on occasions digress widely from the normal and assume a picture closely resembling spindle cell or fibrosarcoma. Some of the cases reported here were first diagnosed as fibrosarcoma, but after more thorough studies, a change in diagnosis to leiomyosarcoma was made. Although cases of fibrosarcoma of the intestine have been reported in the literature, we are nevertheless inclined to believe that they are very rare.

Lymphosarcoma. Generally speaking, these tumors are fairly rapidly growing with a tendency to grow expansively and metastasize early. Their rapid growth is associated with degeneration, necrosis, hemorrhage, and perforation. There were 4 cases of lymphosarcoma in this series, 3 arising in the small in-

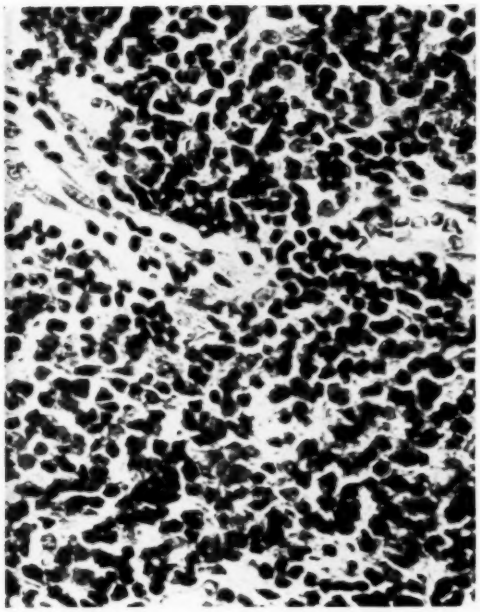

Fig. 3. Photomicrograph of lymphosarcoma. Hematoxylin and eosin stain, ×80.

Fig. 4. Photomicrograph of reticulum cell sarcoma. Hematoxylin and eosin stain, ×80.

testine and 1 in the appendix. The growth was either lobulated or nodular, often soft and friable with tendency to ulcerate and hemorrhage. On section, the tumor appeared homogeneous, grey, pink or yellow in color. In 2 instances the tumor protruded into the lumen of the bowel and was the cause of acute intussusception. The bulk of the growth was usually confined to the mucosa or submucosa, although it may infiltrate the muscular and serous coats.

The close similarity in both architectural pattern and cell morphology of the tumor cells to the lymphoid structure of the intestine is striking and at once suggests a genetic relation. Microscopically, the tumor parenchyma was made up of cells which bore a striking resemblance to the small lymphocyte commonly seen in the lymph follicles of the intestine. The cells were small and round, of about the same size and shape as a lymphocyte. The nucleus was also round, hyperchromatic, and occupied the major portion of the cell substance, while the cytoplasm was scant and formed but a narrow rim surrounding the periphery of the nucleus. Mitotic figures were frequently encountered and designated a high degree of malignancy. The structural arrangement of the cells varied somewhat in different tumors. In places the cells were found crowded together into dense nests with very little intercellular connective tissue stroma to support them; in others, they assumed a more follicular arrangement. The mucosa overlying the tumor was usually intact and well preserved. There was a tendency, however, for the tumor to invade the muscular coat and occasionally also, the serosa (Fig. 3).

Reticulum cell sarcoma. A review of the literature reveals a wide discussion of the genesis of reticulum cell sarcoma. Some investigators classify it as a form of lymphosarcoma, while others consider it to be a type of fibrous connective tissue growth. From the present study it appears to be more closely allied to the lymphosarcomatous group than the fibrous connective tissue forms of neoplasm. The parenchymal cells resemble more closely

Fig. 5. Photomicrograph of neurofibrosarcoma. Note palisading of cells and rosette formation. Hematoxylin and eosin stain, ×80.

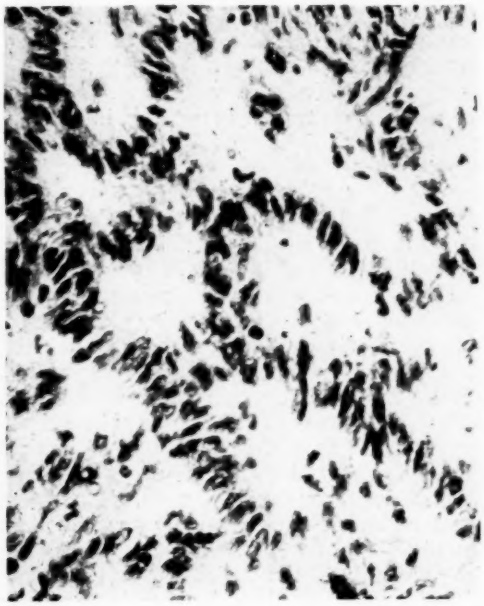

Fig. 6. Higher magnification of Figure 5. Hematoxylin and eosin stain, ×215.

the lymphocyte described in the lymphosarcoma, while the stroma is somewhat more abundant and forms a fine fibrillar or reticular structure.

There were 3 cases of reticulum cell sarcoma, all arising from the small intestine. The structural pattern and cell morphology of the tumors were fairly uniform in all cases. Grossly, the tumors were partly firm and partly soft and friable, arising as a rule in the submucosa and spreading out in all directions. On section, the surface was a homogeneous gray or pink tissue. In one instance the lesion appeared as an ulcerated, cauliflower growth with elevated edges. When this was cut across, a milky fluid escaped from the cut surface. In another case, the tumor invaded the wall of the bowel and caused a tubular narrowing of the lumen for a distance of several inches.

Microscopically, the tumor parenchyma consisted of polygonal cells, varying considerably in size and shape and arranged in groups or nests. The nuclei were irregular in outline and stained deeply. Mitotic figures were frequently encountered, designating a high degree of malignancy. The stroma was moderate in amount and showed a fibrillar or reticular arrangement. The primary site of the tumor was in the submucosa from which it spread out in an irregular fashion toward the muscularis. The mucosa as a rule, was uninvolved and left intact; in one case it was ulcerated. The lymphatic spaces within the wall of the affected bowel were dilated and filled with tumor tissue. The adjoining lymph nodes and fat were also infiltrated with the tumor (Fig. 4).

Neurofibrosarcoma. This type of tumor is very uncommon in the intestinal tract. It occurred in one instance in this series. Its origin can be traced to the tiny nerve fibers in the muscular coat. In the present series the tumor was located in the rectum and consisted of a nodular mass, partly firm and partly soft. On section it presented a granular, yellow gray surface with multiple small hemorrhages.

Microscopically, the tumor was made up of spindle shaped cells of various size. The ar-

rangement of the cells was rather unique in character. In places they formed interlacing bands or pseudorosettes, while in others, they assumed a palisade arrangement. Mitotic figures were rather few in number, indicating a low grade malignancy (Figs. 5 and 6).

ANALYSIS OF STUDY

The clinical picture presented by this group of cases of sarcoma of the intestine merits some detailed description, though in many respects the tumors followed the pattern previously described by many authors. The diagnosis of these tumors is often beset with many difficulties. The early symptoms and signs are by themselves not distinctive and are similar to those associated with other forms of intestinal malignancy. In certain cases the difficulties in arriving at a diagnosis may appear insurmountable. The laboratory tests, although of great value, are found to be by no means infallible.

Practically all the patients presented past histories which may have had bearing on their illness. Analysis of the diagnostic facts relating to these tumors shows that pain is the most constant subjective symptom and is usually described as cramp like in character; it occurred in more than 70 per cent of the present series. Loss of weight was not a frequent complaint and most of the patients appeared well nourished when first examined. Constipation alternating at times with diarrhea was a more frequent complaint.

The disease often pursued a chronic course. The earliest treated patients dated the onset of their illness to 4 to 7 days prior to hospitalization, the latest 2 years, giving the average duration of symptoms of 6 months. When complications set in the onset of symptoms was acute and of short duration. Intestinal obstruction, perforation, and massive hemorrhage are described as the more common forms of complication of the disease.

Physical examination revealed certain significant findings. The majority of the patients showed a secondary anemia which was proportionate in degree to the loss of blood. An abdominal mass was felt in 60 per cent of the patients; the mass was either fixed or freely movable and often tender to palpation.

Among the various forms of nonepithelial malignant tumors of the intestinal tract, the following are most commonly encountered: lymphosarcoma, leiomyosarcoma, reticulum cell sarcoma, fibrosarcoma, spindle cell sarcoma, endothelial sarcoma, and Hodgkin's disease. In the present series four varieties of sarcoma were found and described, namely, lymphosarcoma, leiomyosarcoma, reticulum cell sarcoma, and neurofibrosarcoma. Of these various forms, lymphosarcoma is most common, although in the present series the leiomyosarcoma outnumbered the lymphosarcoma in the ratio of approximately 2 to 1.

Sarcoma of the intestinal tract is as a rule a primary lesion and for a long time confines its growth to the bowel. As the disease progresses, the growth extends to involve the entire thickness and circumference of the intestine and even projects into the lumen or forms a bulge on its exterior, but its tendency is to localize in the bowel and not metastasize. In some instances, however, it tends to extend into adjoining structures, especially into regional lymph nodes. This is particularly true in the cases of the lymphosarcoma and reticulum cell sarcoma. When the mucosa is also involved in the tumor process, ulceration usually follows. These changes are responsible at times for a host of symptoms. Under such circumstances, bleeding may take place from the ulcerated intestinal wall and blood may appear in the stools. Thus, 8 of the patients in this series showed tarry stools which reacted positively to benzidine, and 2 showed gross hemorrhages. Metastasis in the case of the lymphosarcoma and reticulum cell sarcoma takes place by the usual route through the lymph channels. In the case of the other forms of sarcoma, the spread is usually through the venous route.

In view of the present study of these cases and in correlation of the data with the various theories offered on the histogenesis of sarcoma of the intestine, we are inclined to consider these tumors as neoplasms arising from specific constituents of the intestinal wall. Throughout the entire series of cases, the histologic picture presented by the tumors bore a striking morphologic resemblance to the tissue from which they arose. Thus, the leiomyosar-

coma closely resembled the smooth musculature of the intestine, and the lymphosarcoma resembled the lymphoid tissue normally present in the intestinal mucosa. The reticulum cell sarcoma bore a morphologic resemblance to the large, pale staining cells forming the reticulum of the lymph follicles, and there is no doubt as to the origin of this tumor from that structure. It is held by some that this tumor is of fibroblastic origin and by others, that it is of lymphoid origin. From the present study of the morphology of this tumor we are more inclined to believe that it arises from lymphoid tissue. Its structural pattern, site of origin, way of growth, and its spread through the lymph stream rather than the venous channels is most suggestive of lymphoid origin.

The neurofibrosarcoma consists of a mixture of nerve fibers and connective tissue, and there is no doubt, therefore, as to the origin of this tumor from the nerve elements of the intestinal wall.

Diagnosis is invariably late, as it is dependent on the onset of symptoms, and it is well recognized that unfortunately the early symptoms of the sarcoma of the intestine are vague and apt to be confused with those of other lesions in the intestine. There is of necessity a latent period between the earliest tumor formation and the patient's awareness of something amiss, and the duration of this period cannot be established at present. Owing to the low grade of malignancy of some of these growths, by the time the disease has produced symptoms of hemorrhage, obstruction or perforation, it is nearing the terminal stage. In the present series an accurate diagnosis of sarcoma was not made in any of the patients preoperatively, and in many instances a correct interpretation of the lesion was not made until a histologic examination of the specimen was done. In reviewing the cases presented we note several clinical facts. First, in some of the cases of acute involvement, the process was ushered in by the classic syndrome of sudden hemorrhage or sudden intestinal obstruction. In most of the cases the findings were corroborated at operation. Second, the largest group of cases existed in which there was no definite clinical evidence suggestive of the presence of a neoplasm. Roentgen studies of these neoplasms are often helpful in locating the site of origin of these tumors and rendering diagnosis more accurate.

Little need be said of the prognosis in cases of intestinal sarcoma. If the condition is discovered early, long before metastasis has had a chance to occur and complete resection of the lesion is done, a more favorable prognosis may be rendered. This holds particularly true in the more slowly growing tumors such as the leiomyosarcoma and neurofibrosarcoma which grow slowly and metastasize late in the disease. The ideal treatment is complete removal, if possible. When the lesion is discovered late permitting metastasis to occur, the prognosis, as in other malignant tumors, is rather poor.

CONCLUSIONS

Fifteen cases of sarcoma of the small and large intestine are reviewed. In 7 of these the lesion was a leiomyosarcoma, in 4, lymphosarcoma, in 3, reticulum cell sarcoma, and in 1, neurofibrosarcoma.

The diagnosis of sarcoma of the intestinal tract is difficult as it simulates other lesions of the intestine.

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THE TREATMENT OF SEVERE INTRACRANIAL INFECTION BY REPEATED INTRAVENTRICULAR INJECTION OF PENICILLIN

Theoretical and Practical Considerations

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UNDER the impetus provided by the recent war, penicillin has eclipsed the sulfonamides in the results achieved in the treatment of intracranial infections and has provided the physician with a powerful addition to his clinical armamentarium. Combination of both penicillin and sulfonamides has achieved even greater miracles in central nervous system infections. Nevertheless, although the mortality of intracranial infection has thus been greatly reduced in the face of a purely medical attack, there yet remains a group of cases in which the mortality and morbidity is high.

It is in these cases that a combined attack by both physician and surgeon, utilizing all the general and special therapeutic methods at their command in an endeavor to bring the antibacterial agents into the closest possible relationship with the infecting organisms for as long as possible, can materially improve the outlook. Especially is this so in cases of purulent meningitis and ventriculitis, cerebral abscess, posttraumatic intracranial infections, and those intracranial lesions secondary to ear, nose, and throat infections.

A series of these cases, the first of which has been reported elsewhere (Wyke and Vanderfield, 1946) is being studied by the writer; the present paper deals with the theoretical and practical considerations upon which the intraventricular method of treatment is based, together with certain general suggestions regarding the management of severe intracranial infections. Detailed discussion of other cases in the series, and results of special investigations relating to certain problems arising from the present paper, will be reported later.

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Up to the present, no systematic account of the theory and practice of the intraventricular method of administering penicillin has appeared in the literature, although the procedure has been employed sporadically in various centers on numerous occasions.

Before turning to a consideration of the practical technical procedures involved in this method of treatment, certain theoretical considerations upon which its clinical application depends, will be discussed in detail.

FACTORS GOVERNING THE PASSAGE OF PENICILLIN FROM THE BLOOD STREAM INTO THE CEREBROSPINAL FLUID CIRCULATION

It is apparent that penicillin can traverse the blood-brain barrier slowly in two directions under normal conditions, passing either from the blood stream into the cerebrospinal fluid, or vice versa. The evidence for this will be discussed. However, the quantitative aspects of this phenomenon constitute the significant feature, supplying the answer to the problem as to whether an adequate bacteriostatic concentration of the drug can be built up and maintained in the cerebrospinal fluid from doses administered systemically. The temporal relations of the $\frac{\text{serum}}{\text{C. S. F.}}$ penicillin ratio will form the subject of a later communication, but sufficient work has been done to date to suggest certain conclusions.

Early in the therapeutic history of the drug, it was claimed (Abraham and associates, 1941) that intravenous injection of penicillin into cats did not lead to the excretion of significant amounts of the drug into the cerebrospinal fluid. Later, Rammelkamp and Keefer (1943) were unable to demonstrate any penicillin in the cerebrospinal fluid of man following single

intravenous doses of 10,000 to 20,000 Oxford units. The same result was reported in a case receiving penicillin by the continuous intravenous drip method at the rate of 2,500 to 3,000 units per hour, even after 24 hours of such therapy. In addition, Fleming (1943), comparing blood and cerebrospinal fluid concentrations of penicillin during intramuscular administration, concluded that excretion of the drug into the cerebrospinal fluid was negligible. Similar observations were reported by Keefer and associates (1943), Struble and Bellows (1944) and Dolphin (1944).

Officer, Loewenthal, and Perry (1944) stated that their experience was the same, with both intramuscular and intravenous injections of the drug. They cited one case in particular in which no penicillin was detectable in the cerebrospinal fluid although the patient received 200,000 units of penicillin intravenously. Herrell, Nichols and Heilman (1944) were also unable to demonstrate penicillin in the cerebrospinal fluid of 4 patients with meningitis receiving 100,000 units per day, by continuous intravenous infusion.

Notwithstanding these reports to the contrary, Rosenberg and Sylvester (1944) claimed that penicillin was excreted into the cerebrospinal fluid in bacteriostatically adequate amounts. They reported 8 cases of meningitis, in which patients were treated with intramuscular or intravenous penicillin in doses of 20,000 to 40,000 Oxford units every 3 hours, whose cerebrospinal fluid contained the drug in concentrations of 0.03 to 0.35 unit per cubic centimeter within 60 to 140 minutes.

However, these authors noted, as have others (e.g. Rammelkamp and Keefer, 1943; Smith, Duthie and Cairns, 1946) that both absorption and excretion of the drug from and into the cerebrospinal fluid are very variable processes in different individuals, especially in the presence of meningitic inflammation. The difference in the absorption rate in patients with normal and inflamed meninges is well seen in Figure 1, based on results obtained in the writer's series of cases, in which it is apparent that absorption from the subarachnoid space is about twice as rapid in cases with meningitis as in those without. Similar results have been obtained by Cairns and his colleagues.

That the processes of excretion from the blood into the cerebrospinal fluid are also accelerated in meningeal infections is apparent from the work of Walker and Johnson (1945). These workers demonstrated that, whereas under normal conditions in the meninges little penicillin enters the cerebrospinal fluid from the blood, when the meninges are irritated by bacterial, chemical or physical agents excretion is much increased. In normal monkeys with blood penicillin levels of 23.5 to 32.5 units per cubic centimeter no penicillin could be detected in the cerebrospinal fluid. But after inducing a meningeal reaction by concussing the animal or by intrathecal injection of air or bacteria, 0.17 to 0.78 units per cubic centimeter were demonstrated in the spinal fluid. They noted that in man, systemic (intramuscular) injection of penicillin 6 to 8 hours after performance of pneumoencephalography resulted in detectable amounts of penicillin being present in the cerebrospinal fluid within an hour.

Further weight is led to Walker and Johnson's hypothesis by recent work of Lange, Schwimmer and Boyd (1946). These authors demonstrated a three to fourfold increase in permeability of the capillaries in the meninges and choroid plexuses of patients with meningitis. They gave each of their cases an intravenous injection of 5 cubic centimeters of a 5 per cent solution of fluorescein, and estimated the fluorescein content of the cerebrospinal fluid and blood 1 hour later. Using this method it was found that in normal people the fluorescein content of the cerebrospinal fluid does not exceed 300 γ per 1000 cubic centimeters, whereas in the presence of meningitis there is an increase of up to 1000 γ per 1000 cubic centimeters. They also noted that waning of the meningeal inflammation was accompanied by a diminution in the fluorescein excretion, and a rise in the $\frac{\text{plasms}}{\text{C. S. F.}}$ fluorescein ratio. Similar conclusions regarding the increased permeability of inflamed meninges are reported by Kinsman and d'Alonzo (1946).

These results lend weight to the claim of Rosenberg and Sylvester that penicillin may be excreted into the cerebrospinal fluid in cases of meningitis, but for clinical purposes it

must again be emphasized that, although excretion is rapid, absorption from the cerebrospinal fluid is also greatly accelerated (Fig. 1.). Attention should be drawn in this regard to the brief period of Rosenberg and Sylvester's observations (1 to 1½ hours), in the light of data to be discussed.

In this connection the observations of Cooke and Goldring (1945) are of interest. They studied the blood and spinal fluid levels of penicillin in 8 children, ranging in age from 4 to 34 months, who received relatively large doses of the drug (500 to 5000 units per kilogram of body weight) by the intramuscular route. Five of the children had acute meningeal infections: in 4 of these 0.1 to 0.4 unit per cubic centimeter of penicillin was present in the spinal fluid from 15 minutes to 1 hour after injection. However, most of the drug had disappeared within 1 to 1½ hours after injection. A relevant suggestion is a statement by Katz and associates (1946) that in normal infants the permeability of the blood-brain barrier may be higher than in normal adults. Similar findings were previously reported by Katzenelbogen (1935), who quotes Stern and Peyrat as saying that the resistance of the blood-brain barrier increases with the age of the patient. Cooke and Goldring point out that their results indicate that penicillin is excreted into the cerebrospinal fluid soon after intramuscular injection, at a time when the blood titer is at a maximum. However, the cerebrospinal fluid titer is much below that of the serum, in contradistinction to that in the subcutaneous, peritoneal, pleural, and synovial fluids, in which the penicillin concentration approximates to that in the blood. They suggest that this implies that passage of penicillin from the blood into the cerebrospinal fluid depends upon processes other than simple diffusion, and perhaps involves secretion by the choroid plexuses. The concentration of penicillin in the cerebrospinal fluid, following massive parenteral dosage, is relatively low, and the presence of the drug is transient.

The claims of Rosenberg and Sylvester are denied by many of the English workers with penicillin, prominent among whom are Florey and Jennings (1945) and Smith, Duthie and Cairns (1946). The latter authors were able to detect only traces of penicillin in the cerebrospinal fluid of patients with both normal and inflamed meninges following intravenous injection of 100,000 units of the drug. They showed that, although penicillin may traverse the blood-brain barrier under certain conditions (e.g. meningeal inflammation), the concentration of the drug attainable in the cerebrospinal fluid by systemic administration of even large doses is never adequate to control meningeal infections. Furthermore, under such circumstances organisms in the central nervous system are exposed to sublethal concentrations of the drug, and the dangerous implications of this are obvious.

It has been shown that in many cases of intracranial infection, even after completion of treatment, organisms lurk in the choroid plexuses of the ventricles, although not present elsewhere (Smith, Duthie and Cairns, 1946). Moreover, it has been the writer's experience in a number of cases that, although the lumbar fluid may become relatively clear and sterile after commencing medical treatment, purulent fluid containing many cells and organisms may often be recovered from one or both lateral ventricles. Commonly, this is a sequel to the development of a cerebrospinal fluid block and was a feature of a case reported elsewhere (119).

In view of these experiences, certain facts relating to the mode of action of penicillin are relevant to the discussion. It now appears that penicillin possesses both bacteriostatic and bactericidal powers, depending, to a certain extent, upon the local concentration of the drug. Primarily, it is a bacteriostatic agent, acting by inhibiting cell division, and being most effective when multiplication of organisms is taking place (Gardner, 1940; Hobby, Meyer and Chaffee, 1942). However, under certain circumstances, penicillin may act as a bactericide (Officer, Loewenthal, Perry, 1944; Rantz and Kirby, 1944; Garrod, 1945), completely destroying those organisms with which it is brought into relation in sufficient concentration, for sufficient time. The actual mode of action also depends to a certain extent upon the relative proportions of the fractions G, X, K, and F present in the particular preparation of penicillin.

As soon as the influence of the drug is removed, multiplication may recommence, if the organisms have not been destroyed by the intrinsic antibacterial agents of the body (leucocytes and antibodies) or by penicillin present locally in bactericidal concentration. Penicillin must be kept continually in contact with all infected tissues (e.g. meninges and choroid plexuses), in adequate concentration, until the natural body defenses have had time to deal with the infection (Florey and Jennings, 1945); and this cannot be achieved unless the drug is introduced directly into the cerebrospinal fluid.

For such reasons it is essential that adequate concentrations of penicillin should be maintained locally (i.e. in the cerebrospinal fluid, especially within the ventricles) for sufficient time, in order to achieve complete destruction of the infecting organisms. This is particularly important in view of the fact that some organisms may become penicillin resistant although remaining pathogenic, when exposed to sublethal doses of the drug (Abraham and associates, 1941; Rammelkamp and Maxon, 1942; McKee and Houck, 1943; Officer, Loewenthal, and Perry, 1944; North and Christie, 1946). It is therefore important to determine the critical lethal concentration of penicillin for each of the organisms infecting the central nervous system. The relevant figures in this regard are given by Rammelkamp and Keefer (1943) and Walker and Johnson (1945).

It should be pointed out, however, that their results represent minimal concentrations, determined *in vitro*. Other modifying factors are present under clinical conditions which tend to make the effective penicillin levels a little higher. These factors are considered elsewhere in the present paper.

It cannot be denied that clinical cure of meningitis has sometimes resulted in patients receiving penicillin by the systemic route alone (e.g. Price and Hodges, 1944; Dawson and Hobby, 1944; Tee, 1945; Milroy and Hughes 1945). However, such cases provide no index of the efficacy of penicillin excretion into the cerebrospinal fluid, as other factors are involved. Most of these patients receive, or have received sulfonamide drugs as well as penicillin, at some stage of their treatment; and these drugs (especially sulfadiazine) are capable of sterilizing the cerebrospinal fluid in infections with susceptible organisms. In addition, many cases of intracranial infection, especially those of meningococcal or pneumococcal origin, have a concomitant bacteriemia of intermittent type. Possibly the action of penicillin upon the systemic component of the infection may be partly responsible for the clinical improvement.

During the last 12 months further studies have appeared, dealing with the passage of penicillin from the blood stream into the subarachnoid space; and these support the earlier findings.

McDermott and Nelson (1945) have examined the problem with reference to the treatment of neurosyphilis with penicillin. In 70 patients they were unable to demonstrate significant amounts of penicillin in the cerebrospinal fluid by 4 different methods of assay, following parenteral dosage of penicillin in varying amounts. The results were unaffected by the presence or absence of neurosyphilis in the patients. Following intramuscular injections of 300,000 to 500,000 units of penicillin, only 0.02 unit per cubic centimeter were demonstrable in the cerebrospinal fluid three to four hours later.

Following the work of Walker and Johnson (1945) cited earlier, Dumoff-Stanley and her colleagues (1946) examined the penicillin excretion into the cerebrospinal fluid in 14 patients, 6 of whom had intracranial infections. During intramuscular or intravenous injection of the drug, penicillin was detected in the cerebrospinal fluid of 6 patients, 4 of whom had meningitis. The authors found that in 2 babies penicillin was detectable in the ventricular fluid, while absent in that in the subarachnoid space. In another case the drug was present only in the last few cubic centimeters of 120 cubic centimeters of fluid withdrawn during pneumoencephalography. They conclude from these facts that the passage of penicillin from the blood stream into the cerebrospinal fluid occurs primarily through the choroid plexuses, as previously suggested by Cooke and Goldring (1945). They also found a serum concentration of at least 0.625 unit per

cubic centimeter to be the minimal blood level for adequate transchoroidal excretion of penicillin, and that no patient with penicillin in his blood stream for less than 12 hours attained therapeutically effective concentrations of the drug in the cerebrospinal fluid. They therefore conclude that systemic administration of penicillin in the usual doses will not consistently yield measurable concentrations of penicillin in the cerebrospinal fluid.

More recently, Schwemlein and associates (1946) reported that they were unable to detect significant concentrations of penicillin in the cerebrospinal fluid of patients with syphilis following intramuscular doses of 20,000, 40,000, and 200,000 units. However, following massive intravenous administration by the continuous drip method, effective levels were attained in cases of early syphilis. Their series consisted of 167 patients with primary or secondary syphilis, each of whom received 10 to 25 million units of penicillin given by continuous drip for 24 hours. Their results reveal that continuous intravenous administration of 10 to 20 million units produces bacteriostatically adequate concentrations in the cerebrospinal fluid. Use of such large doses in this manner is attended by certain reactions, which the authors list as thrombophlebitis, nausea, vomiting and azotemia; there were no meningeal reactions. Similarly, Kaplan and associates (1946) were unable to detect any significant excretion of penicillin into the cerebrospinal fluid following intramuscular administration in neurosyphilis.

A study of the reports which have been discussed leaves the impression that the efficacy with which penicillin traverses the blood-brain barrier under normal and abnormal conditions remains to be determined. It appears that the Atlantic Ocean separates two schools of thought in this matter. On the one hand is the English school, headed by Florey and Cairns, and including the original workers with the drug, which denies the therapeutic efficiency of systemically administered penicillin in cases of severe intracranial infection, and recommends direct introduction of the drug into the cerebrospinal fluid. On the other is the American school, many members of which question the rationale of this method of therapy (see Reese, 1945) and plead for parenteral administration only. However, dissentient voices are heard in both groups.

In view of the unsettled state of the problem, it is apparent that definitive studies dealing with the time relations between serum and cerebrospinal fluid levels of penicillin under normal and abnormal intracranial conditions should be undertaken. Such investigations are at present being pursued in Sydney, and the results will be reported in a subsequent communication. However, in spite of a few reports to the contrary, it would appear justifiable, on the present available evidence, to conclude that therapeutically adequate concentrations of penicillin cannot be maintained within the cranial cavity by intermittent or continuous systemic administration of the drug in reasonable doses.

For this reason the writer believes that in all cases of intracranial infection with susceptible organisms, except perhaps those of comparatively benign type, penicillin should be introduced directly into the cerebrospinal fluid circulation at repeated intervals, in addition to other therapeutic measures that may be undertaken. In severe cases the indications of which are outlined later, such administration is preferably into the lateral ventricles of the brain, with or without intraspinal administration as well.

DEVELOPMENT OF THE PROCEDURE OF DIRECT INTRODUCTION OF PENICILLIN INTO THE CEREBROSPINAL FLUID

Penicillin was first introduced directly into the cerebrospinal fluid of human patients in 1942, by Fleming (1943). His patient was a moribund case of streptococcal meningitis, and the lumbar route was employed. Florey and Florey (1943) next reported a case of meningitis in which patient was treated with penicillin administered by the cisternal route; and in the same year Smith, Duthie, and Cairns (1943) and Barker (1943) described cases of pneumococcal meningitis successfully treated by intrathecal injections of penicillin. A further 23 cases of pneumococcal meningitis, some of whom received intrathecal injections of penicillin, were reported by Rammelkamp and Keefer (1943). Other cases of meningitis

in which patients were treated in this manner were recorded by Keefer and associates (1943).

Following these accounts, Dolphin (1944) reported 3 cases of suppurative meningitis treated with intraspinal and intraventricular penicillin. This, together with an account by McCune and Evans (1944), constitutes the first reference to use of the intraventricular route for the administration of penicillin in intracranial infection.

Reports of a further 16 cases of meningitis receiving daily intrathecal or intraventricular injections of penicillin were presented by Cairns, Duthie, Lewis and Smith (1944). Their cases were all patients suffering from pneumococcal meningitis; there were 4 deaths in the series. The penicillin was injected into both spine and ventricles in amounts of 4 to 10 cubic centimeters of a solution diluted in isotonic saline so that the concentration was 250 to 500 Oxford units per cubic centimeter. The average single dose into ventricles or spine was 3000 to 4000 units. Following injection of such a dose into the lateral ventricles, penicillin was soon demonstrable in the lumbar fluid, a concentration of 0.4 unit per cubic centimeter (ten times that necessary to inhibit the growth of a sensitive pneumococcus) being present after 24 hours.

The fall in concentration and the relation between ventricular and spinal fluid levels of penicillin at successive intervals are seen in Figure 2, based on figures from the writer's series of cases. It is apparent that the intraventricular concentration falls off in an exponential manner, half concentration being attained in about 5 hours. Complete disappearance of the penicillin in the ventricles results in about 24 hours. The level of penicillin in the lumbar fluid does not parallel that in the ventricles, but pursues a more rectangular course. It must be pointed out, however, that these values are merely averages, the limits of variation in different patients being relatively wide. However, they agree closely with the results of Cairns and his colleagues.

Results similar to those described were noted by Officer, Loewenthal, and Perry (1944). The lumbar fluid of one of their cases contained 0.4 unit per cubic centimeter, 14 hours after injection of 10,000 Oxford units of penicillin into the right lateral ventricle. Penicillin introduced into the lateral ventricles soon spreads, in the absence of a cerebrospinal fluid block, to all parts of the subarachnoid space (Cairns and associates, 1944; Cairns, 1945), and thence slowly into the blood stream (Rammelkamp and Keefer, 1943), whence it is excreted into the urine and bile (Rammelkamp and Bradley, 1943) and perhaps, in minute amounts, back into the cerebrospinal fluid (see Fig. 1).

Further cases of intracranial infection treated with penicillin administered into the spinal theca or cisterna magna were reported by Pilcher and Meacham (1943, 1944); Miller and Ross (1944); Officer, Loewenthal, and Perry (1944); Waring and Smith (1944); Meads and associates (1944); Rosenberg and Arling (1944); Turner (1944); Evans (1944); Vickery and Dey (1944); Herrell, Nichols and Heilman (1944); Worth (1945); Baird (1945); Appelbaum and Nelson (1945); Sweet and associates (1945); Hall and associates (1946); and Naffziger and associates (1946). All of these workers recorded clinical results far better than any achieved previously, and expressed belief in the efficacy of the direct method of treatment.

Later, Johnson and Walker (1945), successfully treated a patient with postoperative meningitis and ventriculitis due to the Staphylococcus albus by direct injections of penicillin into the ventricles through a rubber catheter fixed *in situ*. Baird (1945) used the ventricular route with success in a case of pneumococcal meningitis. Direct injection of penicillin into the ventricles in cases of meningitis was also recommended by Florey and Jennings (1945). In the same year further cases were described in which the intraventricular route was employed for the administration of penicillin Cases of primary meningitis, as well as intracranial infections secondary to war wounds of the skull and brain, which received this method of treatment, were noted by Rewell (1945), Haynes (1945), and Rowe and Turner (1945).

During the present year, additional reports of the use of the ventricular route in the treatment of infected brain wounds, especially transventricular lesions, have appeared as the result of experiences in the recent war. Further

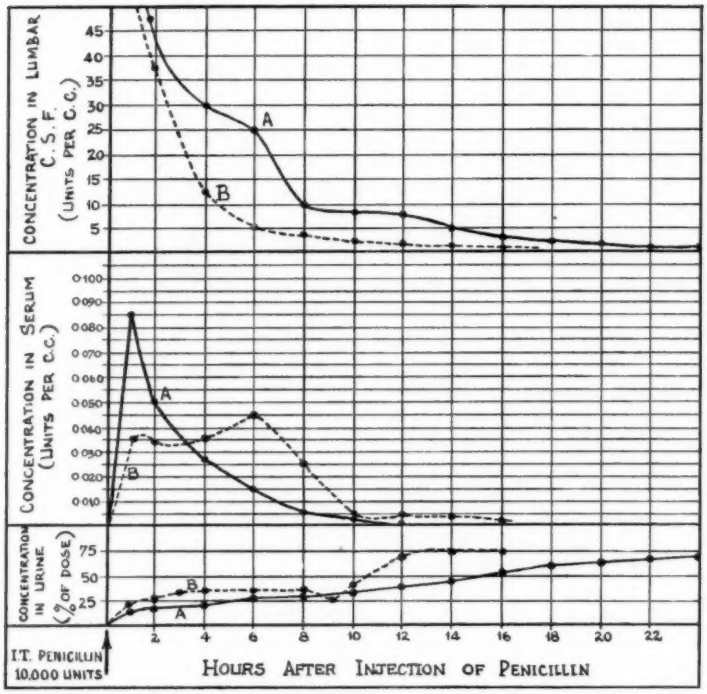

Fig. 1. Quantitative features of the absorption of penicillin from the cerebrospinal fluid following intrathecal injection of 10,000 units, in cases without (*A*) and with (*B*) meningitis. The upper graph represents the exponential decrease in concentration in the spinal fluid. The middle graph indicates the concurrent changes in serum concentration, while the lower one indicates the gradual excretion into the urine as the cerebrospinal fluid and serum concentrations fall.

cases of primary meningitis treated in this manner have also been recorded. Examples of these cases, and the excellent results obtained, are noted in papers by Hudson and associates (1946), Webster, Schneider, and Lofstrom (1946), Smith, Duthie, and Cairns (1946), Turner (1946), Liras (1946), Jepson and Whitty (1946), Miller and Delohery (1946), and Wyke and Vanderfield (1946).

COMPLICATIONS OF DIRECT INTRODUCTION OF PENICILLIN INTO THE CEREBROSPINAL FLUID CIRCULATION

Ever since the introduction of penicillin as an addition to the clinical armamentarium in the attack on intracranial infection, debate has arisen as to possible toxic effects of the drug upon nervous tissue, when in direct contact with it. For obvious reasons, consideration of this question is important in relation to any attempt at direct introduction of penicillin into the cerebrospinal fluid, especially into that within the cerebral ventricles. The evidence bearing on this problem will therefore be reviewed in detail.

The earlier workers with crude penicillin made no mention of reactions to intrathecal or intracisternal injection of the drug, but cases so treated were sporadic and the doses employed were relatively small. Early in the penicillin era it was suggested that the calcium salt of the drug was more toxic to the tissues than was the sodium salt (Florey and Jennings, 1942), but subsequent experience has not substantiated this (Herrell and Nichols, 1943; Györgi and Elmes, 1944; Florey and Jennings,

1945). The elimination of impurities present in the earlier preparations may be partly responsible for this *volte face* (Berger, 1945; Committee on Medical Research, 1946). Whatever be the reason, it should be pointed out that in all the cases studied in Sydney by the writer the patients received intrathecal or intraventricular or both injections of the calcium salt of penicillin, as prepared by the Commonwealth of Australia Serum Laboratories, and no toxic reactions were manifest.

In 1941 it was shown (Abraham and associates, 1941) that intracisternal injection of penicillin in the rabbit produced no histological or functional disturbances. Following this report Florey and Florey (1943), Fleming (1943), Smith, Duthie, and Cairns (1943), and Cairns and associates (1944) employed intrathecal or intracisternal injections of up to 4000 Oxford units in man, without ill effects. Officer, Loewenthal, and Perry (1944) demonstrated the absence of toxic reactions to penicillin in the delicate tissues of the chick embryo, and administered the drug intrathecally to human patients in repeated doses of 15,000 units, without complications.

As larger doses were employed, dissentient reports appeared in the literature, especially in the United States. Pilcher and Meacham (1943) and Rammelkamp and Keefer (1944) claimed that intrathecal injection of penicillin produced irritative changes in the meninges. In a brochure published by Merck and Co. (1943) headache, vomiting, pleocytosis, and increase in pressure in the cerebrospinal fluid were noted as following intrathecal injection of 10,000 Oxford units of penicillin.

In England, Cairns and associates (1944) described systemic reactions following intraventricular injection of 3000 to 4000 units of penicillin. These manifestations consisted of a patchy erythema, sweating, pilomotor activity, and vomiting, lasting some 10 to 20 minutes after injection. However, they pointed out that the preparation used was impure, containing at most only 50 per cent of pure penicillin. Subsequently, Johnson and Walker (1945) described a case of a 22 month old baby in which intraventricular injection of 50,000 units of penicillin diluted in 5 cubic centimeters of normal saline was followed by unconsciousness in 1 hour, and, in 3 hours, by generalized convulsions. These authors believed the effect to be the result of an individual idiosyncrasy to the drug, and mentioned that they had injected up to 100,000 units of penicillin into the ventricles on other occasions without mishap. Idiosyncratic reactions to penicillin given by other routes have been noted (Pyle and Rattner, 1944; Vickers, 1946) although they are rare.

Johnson and Walker's explanation may well be correct, for the writer has repeatedly injected 50,000 units of undiluted penicillin solution (containing 10,000 units per c.c.) into the lateral ventricles of numbers of patients, including two babies under the age of 1 year, without the production of fits or other reactions. Miller and Delohery (1946) also make no mention of toxic reactions following intraventricular injection of 100,000 units of penicillin at repeated intervals. However, Smith, Duthie, and Cairns (1946) mention 4 cases in which signs of lumbar meningeal and cord damage appeared following intrathecal injection of 100,000 units, and Walker and Johnson (1945) had previously noted another. Siegal (1945) made a similar claim in the case of a patient receiving daily intrathecal injections of 40,000 units of penicillin, but in this instance the sequelae were probably more the result of the disease than of the intraspinal therapy. In monkeys, Walker and Johnson (1945) described perianal paresthesia following intrathecal injection of 20,000 units of penicillin diluted in 1 cubic centimeter of normal saline. The effects were apparent in 10 minutes and lasted up to 1 hour. These authors suggested that lumbar radiculitis and arachnoiditis were possible complications of massive intraspinal therapy with penicillin. They also noted the occurrence of a transient increase in cell count in the spinal fluid following intrathecal injection of penicillin. The same phenomenon (i.e. pleocytosis) has been encountered twice by the writer following intraventricular injection of the drug. This reactive pleocytosis appears to be far less common now than in the earlier days of the penicillin era, perhaps because of the increased purity of the available preparations; moreover, it appears that this reaction may not be due primarily to the penicillin (43).

That the purity of the penicillin preparation employed may be a relevant factor in the production of toxic reactions to direct introduction of the drug into the cerebrospinal fluid was first suggested by Cairns and associates (1944). Previously, Florey and Jennings (1942) had claimed that toxic reactions to systemic administration of the drug became less frequent with increasing purity of the preparation. This has been borne out by subsequent experience (Hamre and associates, 1943, 1944; Robinson, 1943; Berger 1945; Committee of Medical Research, 1946) and by the writer, in respect of direct introduction of the drug into the cerebrospinal fluid.

These remarks, however, do not appear to apply to the occurrence of fits, which are unrelated to the purity of the preparation. Arising out of an earlier experience (Johnson and Walker, 1945), Walker and his colleagues suggested the possibility of a "convulsive factor" being present in penicillin and began a series of experimental investigations upon the problem. The possibility of fits or other cerebral reactions to intraventricular injection of penicillin has also been raised by Smith, Duthie, and Cairns (1946).

Walker and Johnson (1945) and Johnson and associates (1946) have described the occurrence of convulsions in cats, dogs, monkeys, and human patients in whom penicillin solution was injected into the cortex or subdural space. Similar reactions following intracisternal injection of 50,000 units were noted by Neymann and associates (1945) in two human patients, who eventually became comatose and died. Walker and Johnson have shown that the convulsive effects vary with different animal species and appear to be governed more by the absolute amount of penicillin injected than by the concentration in which it is employed.

These authors obtained electroencephalographic evidence of cortical disturbance following direct application of penicillin to the brain in adequate amounts. In man, injection of 5000 Oxford units of penicillin into the occipital cortex caused no clinical or electroencephalographic changes; but spike discharges, without clinical disturbances, were initiated by injection of 20,000 units of the drug into the same region. Intracortical injection in the precentral motor region led to twitching of the face and hands for 3 hours. These results were not produced by injection of similar amounts of saline into the same areas of the brain. Verification of these claims has been obtained by Walker, Johnson, and Kollros (1946), Johnson and associates (1946), and Walker and associates (1946), and the question of a convulsive component in penicillin is discussed in a recent issue of the *Lancet* (1946).

As a result of the work of Walker and his colleagues, it appears that adequate concentrations of penicillin are capable of initiating convulsive phenomena in the brains of vertebrates. Apparently there is a "convulsive threshold," in terms of the amount of penicillin necessary to fire off epileptiform discharges in the cerebral cortex, which differs with different animal species. This threshold may be species specific for any one group of animals, and appears to be related to the phylogenetic status of the brain of the animal concerned. Thus it is highest in man (10,000 to 20,000 units per c.c.) and lower in monkeys (500 units per c.c.) and in dogs. Moreover, the convulsive threshold varies also in different cortical areas, being directly related to the electrical excitability of the various portions of the brain; the motor cortex has the lowest convulsive threshold (about 10,000 units per c.c. in man), and responds more readily to the convulsive factor in penicillin than do other areas of the brain. In addition, the convulsive and antibiotic principles in preparations of penicillin appear to be mutually interdependent, as Walker and Johnson (1945) have demonstrated that their inactivation proceeds *pari passu*, when the preparation is subjected to aging, boiling, drying, acidification, and alcoholization. It may be that both the antibiotic activity and the production of convulsive phenomena are properties of one or more active principles of penicillin, and perhaps of all the bactericides of fungus origin, for it has been shown that they are not produced by penicillin alone.

Other antibiotic fungus derivatives, such as streptomycin, streptothricin, actinomycin and clavicin, produce irritant and convulsive effects when applied directly to the brain in suf-

ficient concentration (Buggs and associates, 1946; Forgacs and associates, 1946; Johnson and associates, 1946; Walker and associates, 1946). In the case of the latter 3 these disturbances are of such an order as to preclude their local application to the central nervous system. Among the antibiotics at present available, penicillin and streptomycin appear to have the least active convulsive components in relation to their antibacterial activity. With these two agents there is a wide difference between the convulsive threshold and the effective therapeutic level (Johnson et al., 1946).

Pathological evidence of irritant effects of penicillin on brain tissue has been obtained by Russell and Beck (1945). These workers found that functional and histological changes (hemorrhage and necrosis) occurred in the brains of rabbits in which concentrated penicillin solution (10,000 units per c.c.) was applied directly to the cerebral cortex. Similar changes are also described in the brains of monkeys by Johnson and colleagues (1946).

The result of these investigations, especially in the United States, has been to stimulate suspicion of the procedure of direct introduction of penicillin into the cerebrospinal fluid of human patients. The culmination of these fears is expressed in an editorial in the recent *Year Book of Neurology* in which Hans Reese (1945) seriously questions the rationale of such methods of treatment and advises against their use.

However, the conclusion to be drawn from all these reports is not that they contraindicate intraspinal or intraventricular administration of penicillin, but that there is a limiting amount of the drug, varying with different animal species, which may safely be brought into direct relationship with the central nervous tissue. This appears to be about 10,000 Oxford units per cubic centimeter in man, when applied directly to the motor cortex. Provided a local concentration of this degree is not exceeded in the intraventricular fluid, the direct introduction of penicillin into the lateral ventricles of the brain is an entirely safe proceeding.

As has been previously pointed out, the writer has repeatedly injected 50,000 units of penicillin into the lateral ventricles of patients varying in age from 2 months to 53 years, without untoward effects. This dose is administered as 5 cubic centimeters of a solution of the calcium salt of penicillin, in a concentration of 10,000 units per cubic centimeter.

That such doses are well within the limits of safety in the case of human patients is well borne out by the experiences of many neurosurgeons in the recent war. Accounts based on observations of hundreds of cases of posttraumatic intracranial infection, from many theaters of war, in which penicillin was introduced into the ventricles, subdural space, or into brain cavities, or applied directly to the cerebral cortex, testify to the efficacy and safety of intraventricular injection of penicillin. Reports by Florey and Cairns (1944), Cairns (1945), Worth (1945), Haynes (1945), Rowe and Turner (1945), Webster, Schneider, and Lofstrom (1946), Turner (1946), Martin and Campbell (1946), Weaver and Frisham (1946), Smith, Duthie, and Cairns (1946), and Jepson and Whitty (1946) provide ample evidence of the value of the intraventricular method of treatment in cases both of primary and of secondary intracranial infections.

CLINICAL INDICATIONS FOR REPEATED INTRODUCTION OF PENICILLIN INTO THE LATERAL VENTRICLES OF THE BRAIN

Officer, Loewenthal, and Perry (1944) proposed the following principles to be observed in treating any disease with penicillin: (1) the infecting organism must be sensitive to the action of penicillin; (2) the active principle must be brought into contact with the organisms in sufficient concentration; (3) abscesses must be drained; (4) dead tissue must be removed. These postulates are valid for intracranial infections, as well as for infections elsewhere. With them in mind, and in the light of the previous theoretical discussion, the following criteria are suggested as guides to the selection of cases suitable for treatment by repeated intraventricular injections of penicillin. Presupposed, of course, is the availability of personnel and facilities for carrying out the necessary procedures.

1. Any case of intracranial infection with a penicillin sensitive organism, in which the

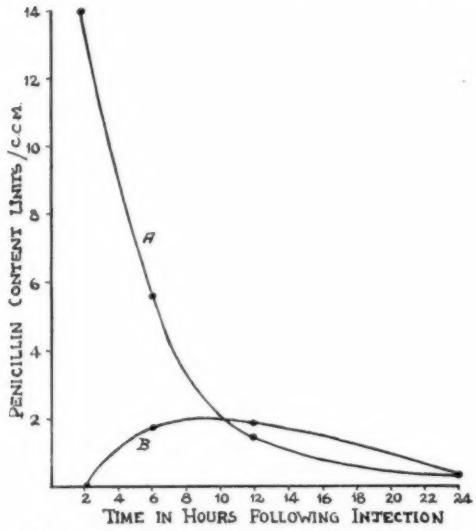

Fig. 2. The relative concentrations of penicillin in the ventricular and spinal cerebrospinal fluid following intraventricular injection of 10,000 units. The ventricular concentration falls off in an exponential manner, while that in the lumbar fluid pursues a more rectangular course. *A*, ventricular fluid, *B*, lumbar fluid.

cerebrospinal fluid obtained at the initial lumbar puncture is obviously purulent, with a cell count of over 600 polymorphonuclear leucocytes per cubic millimeter, and at a pressure greater than 250 millimeters of water.

In all cases, an essential preliminary to the institution of the intraventricular method of treatment is bacteriological identification of the infecting organism. Whenever possible, this should be supplemented by quantitative estimation of its penicillin sensitivity. Even if identification of the organism and determination of its sensitivity has already been performed as a corollary to other methods of therapy, the assay should be repeated either at lumbar or ventricular puncture, at the outset of intraventricular penicillin therapy, and in some cases, during treatment by this method. This is especially important in cases in which the intraventricular method of treatment is indicated by failure to respond to medical therapy with sulfonamides or penicillin, as the infecting organism may belong to a penicillin insensitive group, or have developed resistance to the drug during treatment. In addition, some strains of organisms usually considered unresponsive to penicillin may be sensitive to its action. An example of this is Pittman's (1931, 1933) S (b) strain of Hemophilus influenzae, which has recently been shown to be sensitive to the action of penicillin (27) to about the same degree as a standard staphylococcus. Forgacs and his colleagues have successfully treated with penicillin a patient with influenzal meningitis, due to this organism.

2. Cases of intracranial infection with an organism of high virulence, or with organisms which are only sensitive to relatively high concentrations of penicillin.

The previous discussion has shown that high concentrations of penicillin cannot be attained or maintained in the cerebrospinal fluid within the cranium by systemic administration of the drug. Therefore, in order to maintain high local concentrations of penicillin in both ventricles and subarachnoid space, it should be introduced directly into the cerebrospinal fluid circulation by intraspinal and intraventricular injection. This is especially important in babies and young children, and may be performed with ease by fontanel puncture if the bregma has not completely closed. Any case of pneumococcal meningitis in infants is a special indication for the early use of the intraventricular route, and it should be employed more often in such cases, in view of the relative frequency and high mortality of this disease; for in 642 cases of meningitis in children, Lindsay, Rice and Selinger (1940) found 11.4 per cent to be due to pneumococci. Institution of repeated intraventricular injections of penicillin may be a life-saving measure in these cases. An example of this is provided by two babies suffering from pneumococcal meningitis, recently seen by the writer, who were considered to be moribund, and in whom systemic and intraspinal therapy had failed to produce any response. Following 6 daily intraventricular injections each of 30,000 units of penicillin, both patients recovered completely.

3. Cases of intracranial infection with a susceptible organism which fail to show early and progressive improvement under medical treatment.

Any case of intracranial infection in which there is not a satisfactory response to systemic

administration of penicillin, or the sulfonamides, or both with or without intraspinal penicillin therapy, should receive intraventricular injections of the drug if the causative organism is penicillin sensitive. Especially does this apply to any comatose patient with intracranial infection who does not recover consciousness within 12 hours of instituting medical treatment, or any case in which papilledema develops during medical treatment.

4. Cases of intracranial infection with a penicillin sensitive organism where a cerebrospinal fluid block is expected or suspected.

Cerebrospinal fluid block may occur anywhere in the ventricular or subarachnoid space in cases of intracranial infection. The common sites are in the posterior cranial fossa, in the sylvian aqueduct, within the foramen of Monro, in relation to the tentorium cerebelli, or in the lumbar theca. It has been suggested that herniation of the cerebellar tonsils through the foramen magnum as a result of the high intracranial pressure is a common cause of block in cases of meningitis (Shalom, 1945). However, this has not been a common feature in the writer's cases; more frequent causes of block are the formation of fibrinous adhesions which obstruct the free flow of cerebrospinal fluid, or compression and displacement of the ventricular system by the development of an intracranial abscess. But in the presence of an intracranial abscess, tonsillar or uncal herniation may become a potent cause of block, especially if frequent lumbar punctures are performed.

The frequency with which cerebrospinal fluid block occurs as a result of adhesion formation depends upon a number of factors, the most important of which will be discussed further

a. The nature of the infecting organism. The development of cerebrospinal fluid block and consequent hydrocephalus appears to be most frequent in pneumococcal infections of the nervous system. It is less frequent with Staphylococcus aureus infections, and still less in those due to meningococci and streptococci. The formation of basal adhesions is a complication of considerable significance in pneumococcal meningitis, as it may lead to death of the patient, even though the cerebrospinal fluid has been completely sterilized. For this reason, the use of agents to prevent formation of the fibrinous adhesions which later become organized into dense fibrous bands is under consideration. Heparin has been suggested as a possible solution to this problem (Signorelli and Friedman, 1945; Turner, 1946); and the writer is at present investigating the possibility of introducing heparin, or a fibrinolysin prepared from blood, into the ventricles along with the penicillin, in an endeavor to prevent the formation of the primary adhesions, which are the result of deposition of fibrin. Later, these become organized (if the patient survives the infection) by fibroblastic permeation from the blood vessels, and then constitute dense secondary adhesions which are intimately blended with the thickened arachnoid. If formation of the primary adhesions can be prevented, the incidence of postinfective hydrocephalus, with its entailed mortality and morbidity, will be reduced to a great extent.

These complications are becoming increasingly frequent as more and more patients survive intracranial infections which in other days were fatal. The use of sulfonamide drugs and penicillin inhibits bacterial activity, but does not check the fibroblasts which proliferate in an attempt to organize the purulent exudate in the subarachnoid space. The result is the formation of adhesions and the conversion of the leptomeninx into a dense scar through which the cerebrospinal fluid cannot circulate. A series of such cases following sulfonamide therapy has been recorded by Bailey (1945); and the writer has seen several examples following treatment of meningitis with penicillin. Investigation of this problem demands urgent attention, because it is of little use sterilizing the cerebrospinal fluid if the patient dies subsequently from the results of an obstructive hydrocephalus. Application of the principles detailed in this paper, and the institution of the intraventricular method of treatment at an early stage, will often help to prevent adhesion and abscess formation; nevertheless, there will still remain a few cases in which adhesions form early in the disease. These cases, perhaps, may be aided by pro-

phylactic utilization of some fibrinolytic agent along with the antibacterial drugs.

b. Antifibrinolytic action of penicillin. Certain organisms which infect the central nervous system possess fibrinolytic properties, which result from the activity of an enzyme complex (fibrinolysin, fibrinase) which they elaborate. Organisms differ in the activity of the fibrinase they produce, fibrinolysis being most active in the case of Staphylococcus pyogenes, less so with Streptococcus pyogenes, and slight with pneumococci and meningococci. Different strains of the same organisms also display differences in fibrinolytic activity, some strains being very active, while others (even in the case of Streptococcus hemolyticus) being almost inactive.

It has been shown by several workers (Rammelkamp, 1942; Wilson, 1943; Fleming, 1943, 1944) that the fibrinolytic activity of Streptococcus hemolyticus is inhibited by penicillin. This has been confirmed by Neter and Will (1944). More recently, Dixon (1945) has demonstrated that fibrinolysis by Staphylococcus pyogenes is also inhibited by penicillin, being completely stopped by *in vitro* concentrations of penicillin of 0.1 unit per cubic centimeter and greatly retarded by 0.05 unit per cubic centimeter. The phenomenon is independent of the presence of serum, and may reasonably be expected to take place in the cerebrospinal fluid. This antifibrinolytic activity appears to be a property of the active principle of the drug, for it is related to the penicillin sensitivity of the organism. With penicillin resistant strains, fibrinolysis is unimpeded by the presence of the drug (Dixon, 1945).

Thus it would appear that, theoretically at least, penicillin is capable of inhibiting the activity of the enzyme responsible for fibrinolysis, elaborated by some of the organisms causing intracranial infections in man. What the relation may be between this discovery and the formation of primary fibrinous adhesions remains to be seen. It will be interesting to determine in this regard, whether, since the introduction of penicillin, the frequency of adhesion formation has shifted its peak from the pneumococcus group toward the staphylococcus-streptococcus group of intracranial infections. Nevertheless, the aforementioned data suggest that the introduction of a fibrinolytic agent, which is not inhibited by penicillin, into the cerebrospinal fluid may be an integral part of the treatment of severe intracranial infections.

Cerebrospinal fluid block occurs long before the development of an abnormal response to the Queckenstedt test. One of the earliest indications of the occurrence of a block in intracranial infection is gradually increasing difficulty in obtaining fluid at lumbar puncture, even though it is not obviously purulent (Shalom, 1945; Smith, Duthie, and Cairns, 1946). Increasing yellowness of the spinal fluid obtained at successive spinal punctures for intrathecal injection is also suggestive of block. Another useful index of a developing block in a patient being treated by intrathecal injection of penicillin is a retardation of the fall off in concentration of the drug in the lumbar fluid. If no block is present, the drug is absorbed and diffuses away, so that its local concentration falls off decrementally in an exponential manner, half concentration being reached in about 4 to 5 hours. Performing serial assays on the lumbar fluid, if a block is suspected (Fig. 3), will often reveal that the concentration curve has lost its exponential character and is tending to plateau formation. This suggests a developing block, and occurs long before there is any detectable change in the Queckenstedt response: the higher the plateau, the more complete the degree of block.

The development of such a phenomenon is a strong indication for treatment by the ventricular route. Cairns and his colleagues (1944) previously stated that development of cerebrospinal fluid block in cases of intracranial infection demands intraventricular injection of penicillin, and with this the writer agrees. at the same time emphasizing the futility of waiting for an impaired Queckenstedt response before making a diagnosis of spinal block.

5. Cases in which cerebral or cerebellar abscess formation is suspected.

This applies particularly to cases of intracranial infection secondary to infective lesions of the ear, nose, or throat. Cairns (1945) pointed out that intrathecal administration alone of penicillin is useless in meningitis

secondary to brain abscess or suppurating extracranial foci.

Cases of posttraumatic infection, especially where ventricular penetration has occurred, also fall into this group.

The formation of a cerebral or cerebellar abscess is an important complication of primary or secondary intracranial infection and must always be considered in cases in which the disease becomes prolonged, or is slow to clear up under medical therapy.

The diagnosis of intracranial abscess may present extreme difficulty and will not be considered in detail in the present paper, except to stress the value of electroencephalography as a diagnostic aid. Not only may the presence of an abscess be revealed by the appearance of an abnormal rhythm (see Fig. 4), but its location may be indicated by employing the procedures of "phase reversal" and "triangulation." However, it should be pointed out in passing that a negative electroencephalographic tracing is not exclusive of abscess formation, and for this reason in all suspected cases ventriculography should be carried out as a confirmatory step, and penicillin injected into the ventricles through the burr holes introduced during the procedure.

Two types of abscesses occur within the cranium: the acute and the chronic (or encapsulated) forms. The recent work of Clovis Vincent (1946), Pennybacker (1945), and Furlow (1945) suggests that the correct treatment of a cerebral abscess is excision after encapsulation has occurred. Direct surgical attack is contraindicated during the preliminary acute phase of abscess formation; but it is during this "expectant" period that penicillin can play an invaluable part, especially if introduced directly into the ventricles as well as by other routes. However, in the early stages of abscess formation cerebral edema is generally marked, and the consequent brain swelling may reduce the ventricles to mere slits, precluding the performance of a ventricular tap. Nevertheless, even if such be the case, the introduction of burr holes will permit irrigation of the cerebral subarachnoid space with penicillin solution. As the abscess becomes walled off, its contents may be aspirated through a burr hole and its cavity filled with

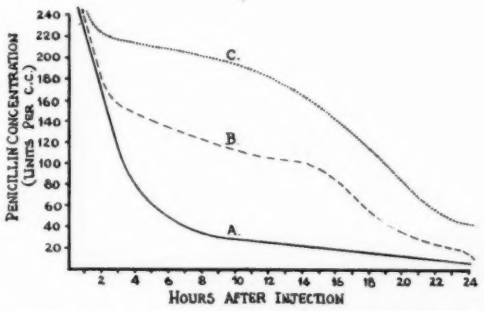

Fig. 3. The course of penicillin concentration in the spinal fluid in cases of meningitis with developing cerebrospinal fluid block, as determined at serial lumbar punctures. As the block develops, the absorption curve loses its exponential character and becomes plateau-like. *A*, The normal curve in cases of meningitis without block, *B*, the curve in cases with an early developing block, *C*, the curve in cases with complete cerebrospinal fluid block.

penicillin solution, and finally, when encapsulated, it may be removed by radical excision, together with any brain tissue that is obviously infected (King, 1924; Fincher, 1946; Sachs, 1946). Excision of the abscess should be immediately preceded by intrathecal and intraventricular injection of penicillin as a prophylactic preoperative measure (q.v.); in some cases marsupialization of the abscess may be carried out.

Electroencephalography again is useful in following the transition of an acute cerebral abscess into the encapsulated form, and the performance of serial electroencephalographic examinations during the waiting period helps to determine when the critical time for operation has been reached.

6. Any case of intracranial infection with streptococci.

Any case in which streptococci are identified in the cerebrospinal fluid is a potential case of cerebral abscess. Smith, Duthie, and Cairns (1946) found 6 of 8 consecutive cases of streptococcal meningitis to have a cerebral abscess. Streptococci in the cerebrospinal fluid therefore demand ventricular puncture and ventriculography, and serial electroencephalograms where possible. If an abscess is found, direct introduction of penicillin into the ventricles and abscess cavity along the lines here indicated is essential. Even if no abscess is found, repeated intraventricular in-

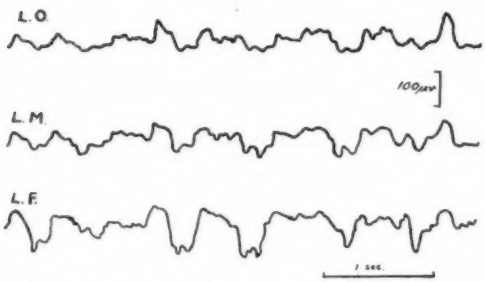

Fig. 4. An electroencephalogram showing high voltage (125 uv.), slow (2-3 c/sec.) delta waves, such as are seen in the region of cerebral abscess formation, after encapsulation. A tracing of this type is an indication for treatment along the lines described in this paper. The record is taken with monopolar leads in the left frontal, motor, and occipital regions.

jection of penicillin is the best method of treatment in all cases of streptococcal intracranial infection. No patient with streptococcal meningitis should ever be subjected to lumbar puncture twice after it has been identified as such (Smith, Duthie, and Cairns, 1946); the direct ventricular route should immediately be resorted to.

7. Cases in which intracranial operations are to be carried out upon, or in the presence of, potentially infected foci.

Intraventricular injection of penicillin may be used prophylactically as a preoperative procedure, combined, if necessary, with intraspinal administration of the drug. Its use has been already referred to, and Cairns (1945) recommends that a single dose of 4000 to 8000 Oxford units be injected into the ventricles prior to operations for (a) removal of chronic brain abscess, (b) closure of a cerebrospinal fistula, (c) dural repair after orbitofrontal or nasofrontal wounds, or lesions involving the petrous temporal bone. This procedure should be used more often as a preliminary to surgical attack on potentially infected craniocerebral wounds, particularly those in which intracerebral foreign bodies are present. Cerebrospinal rhinorrhea is an especial indication for repeated intraventricular injection of penicillin, which should be commenced immediately the condition is manifest, and continued after its operative treatment.

To conclude this section of the paper, the groups of cases which the author believes to indicate the use of repeated intraventricular injection of penicillin solution are summarized below.

1. Any case of purulent intracranial infection with a penicillin sensitive organism, in which the cerebrospinal fluid pressure and cell counts are high.

2. Any case of intracranial infection with highly virulent or partially penicillin sensitive organisms, especially in children.

3. Any case of intracranial infection with a penicillin sensitive organism which fails to show early and progressive improvement under medical treatment.

4. Any case of intracranial infection with a penicillin sensitive organism in which cerebrospinal fluid block is suspected.

5. Any case in which intracranial abscess formation is suspected.

6. Any case of intracranial infection in which streptococci are found in the cerebrospinal fluid.

7. Any case in which intracranial operations are to be carried out in the presence of potentially infected foci.

8. Any case of head injury that develops cerebrospinal rhinorrhoea.

9. Any case of intracranial infection with a penicillin sensitive organism that develops papilledema.

10. Any comatose case of intracranial infection.

TECHNICAL CONSIDERATIONS.

A. Operative procedure. The initial operation is the same as that for the performance of ventriculography, two burr holes being introduced into the skull, under local anesthesia, either in the parietal or frontal regions. The diploe of the bone is first inspected for evidence of osteomyelitic infection. The dura is seen in the depths of the burr holes, and, after inspection for evidence of epidural infection or abscess formation, is opened with a fine knife and hook. In some cases a subdural empyema may be found overlying the brain (Kubik and Adams, 1943; Spitz and others, 1945). If present, this may be drained, and a fine rubber tube inserted and left *in situ*, through which penicillin may be repeatedly instilled into the subdural space. If epidural

or subdural infection is present, no attempt should be made to needle the ventricle through the infected locus on that side; the opposite side should be utilized.

Brain needles are then passed into the lateral ventricles and the contained fluid is aspirated. A rough qualitative estimate of the intraventricular pressure can be made and compared with that on the opposite side, and the depth at which the ventricle was tapped should be read off from the centimeter scale on the brain needle, as it provides an estimate of the size of the ventricle. A separate needle should be used to tap each ventricle.

The cerebrospinal fluid obtained from each ventricle should be collected in separate sterile containers, and examined individually for volume, turbidity, cell count, protein content and organisms, as this furnishes valuable evidence of the presence or absence of intraventricular block. Further evidence of this, and the patency of the foramen of Monro, may be adduced by washing sterile Ringer's solution through from one ventricle to the other, with both brain needles in place, or by the introduction of methylene blue or filtered gas into one ventricle to see if it will pass to the opposite ventricle. If the fluid obtained from one or both ventricles be turbid, as much of it as possible should be removed, by gentle aspiration with a syringe if necessary, and the ventricles should be washed out with warm sterile Ringer's solution until the returning fluid is clear.

If ventriculography is to be performed, 10 to 20 cubic centimeters of filtered air or oxygen are injected into one (or both) ventricles, the brain needles withdrawn, and the scalp wounds sutured.

The patient remains on the operating table, and the requisite radiographic pictures are taken. This procedure outlines the ventricular system, disclosing any asymmetry or displacement indicative of an abscess, and assists in the diagnosis of intraventricular block.

When satisfactory films have been obtained, the patient, still on the operating table, is returned to the theater, and a fresh brain needle is introduced into each ventricle through the closed scalp wounds. The contained air is aspirated with a syringe, and 3 to 5 cubic centimeters of penicillin solution containing 10,000 units to the cubic centimeter is injected, after dilution with 5 to 10 cubic centimeters of warm saline, if desired, At first, in view of reports of reactions to intrathecal injection of concentrated penicillin solutions (*vid. sup.*) high dilutions were employed for intraventricular injection, of the order of 500 to 1000 units per cubic centimeter; Cairns (1945) recommended 2000 units per cubic centimeter. Now, however, "pure" penicillin solution as supplied by The Commonwealth Serum Laboratories in concentrations of 10,000 units per cubic centimeter is injected without further dilution directly into the ventricles in amounts of up to 5 cubic centimeters; no reactions to this procedure have been seen on the now numerous occasions on which this has been done, even in babies.

B. Postoperative management. On completion of this procedure the patient is returned to bed, after the wound is dressed, and subjected to the usual postoperative routine. A course of systemic sulfadiazine (4 gm. *statim*, then 2 gm. in 4 hours, and 1 gm. fourth hourly thereafter) is begun, if not already commenced. If desired, parenteral administration of sulfonamide may be instituted in the early critical stages. Fluid balance charts are rigorously kept, and fluids given by intragastric or intravenous drip methods on the slightest pretext.

Some of these patients become very restless, or even delirious, following the preliminary operation, and should be restrained with cuffs on legs and arms, and bedrails if necessary. Sedation may be accomplished with repeated intramuscular injections of dial in doses of 1 to 2 cubic centimeters with or without paraldehyde given orally or *per rectum*. In very restless patients, especially if at all dehydrated, a Ryles' tube may be passed and left *in situ* after restraint has been arranged, and through this fluids and paraldehyde may be introduced as required.

In those cases in which there is persistent cerebral edema, especially those that have preoperative papilledema, in which the cerebrospinal fluid pressure often remains high, rectal hypertonic solutions may be given. For

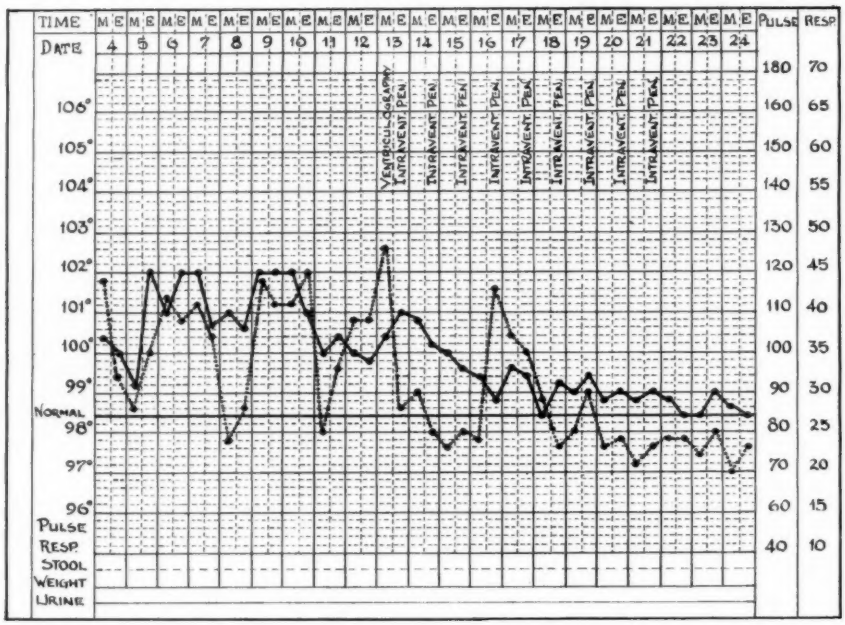

Fig. 5. The clinical chart of a case of purulent meningitis and ventriculitis due to Staphylococcus aureus, treated with 9 daily intraventricular injections of penicillin. The rapid recovery following institution of this method of treatment is well seen. The clinical course prior to this was fluctuating, with a gradual trend for the worse, under a month's medical treatment with sulfonamides and systemic and intrathecal penicillin (from Wyke and Vanderfield, 1946). Pulse, black line, temperature interrupted line.

adults, 6 ounces of magnesium sulfate in 50 per cent solution may be given every 8 to 12 hours and retained. In the case of young children and babies, a rectal hypertonic saline solution is used, prepared in the following manner: 6 drachms of 17½ per cent saline solution are made up to 2 ounces with distilled water, making a sufficiently hypertonic solution which can be given at repeated intervals until the pressure inside the skull returns to normal. A useful indication of cerebral edema, which may be marked postoperatively, is a rapid fall in cerebrospinal fluid pressure after withdrawal of only a small volume of fluid at spinal drainage. If cerebral edema is marked, treatment by spinal drainage is of little avail, and intravenous injections of up to 50 cubic centimeters of hypertonic sucrose should be given.

Each day in the ward a brain needle is introduced into the alternate ventricle (if there is no block), or into both ventricles (if there is an intraventricular block), and the cerebrospinal fluid gently aspirated with a syringe. A cell count and culture is done on each specimen of fluid removed each day for control purposes, and the results graphed, if required. Such a graph (of the pressure and cell count) is a useful index of the efficacy of treatment. After the fluid is removed the ventricle is washed out with warm Ringer's solution, and the penicillin is introduced in the same strength and amount as before. Then the wound is redressed.

A mask should be worn, and sterile gloves donned before commencing the procedure. The patient is postured on his side, with the head flat and slightly flexed, so that the burr hole to be tapped is uppermost. The dressings are then removed with sterile forceps by a nurse, and the field is swabbed with metaphen and covered with a sterile area sheet. The

sutures in the wound are not interfered with, the burr hole being easily found with the point of the brain needle. At the same time lumbar puncture may be performed and penicillin instilled intrathecally, although in most cases this is an unnecessary adjunct.

The procedure detailed is repeated daily until the cerebrospinal fluid pressure falls to normal limits and the fluid becomes clear, the cell count in the ventricular fluid is within normal limits, and the cultures of fluid from each ventricle are sterile on three successive occasions. Considerable improvement is usually apparent within 2 to 4 days, and in most cases not more than 6 to 9 intraventricular injections are required. A typical result from 9 such injections is seen in the clinical chart in Figure 5.

The performance of daily intraventricular injection in the ward is not dangerous, if carried out with the strictest aseptic routine, and has been done many times by the writer without mishap. It should only be performed, however, by an operator skilled in the procedure. Theoretically, the greatest danger is the introduction of contaminant organisms into the cranium that may be penicillin resistant, or penicillinase producers; this has never occurred in any of our cases.

The burr holes should be made well away from the cerebral motor region, in case an intracerebral abscess forms along the track of the needle, leading to gliosis and cerebral scar formation, and the possibility of subsequent epilepsy. This is especially important in children, but may be avoided by using only frontal or parieto-occipital burr holes, and by withdrawing the brain needle on every occasion while actually injecting the penicillin solution. In babies the procedure of ventricular injection may be carried out by puncturing the lateral angles of the superior fontanel with a fine steel needle, but the writer feels that perhaps even in these cases burr holes should be employed, in view of the possibility mentioned above.

SUMMARY

1. The factors controlling the passage of penicillin through the blooddrain barrier are discussed in the light of previous experience and the results obtained by the writer in a series of cases. It is concluded that therapeutically adequate concentrations of penicillin cannot be maintained for sufficient time in the cerebrospinal fluid by systemic administration of the drug.

2. The historical features of the development of the procedure of direct introduction of penicillin into the cerebrospinal fluid are reviewed.

3. Possible complications of the procedure are discussed, and shown to be avoidable and of small significance if proper care is exercised.

4. Certain clinical criteria which the writer considers to indicate repeated intraventricular administration of penicillin are enumerated and discussed.

5. The operative procedures, and post-operative management of cases thus treated, are described.

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THE SIGNIFICANCE OF SKIN RECURRENCES FOLLOWING RADICAL MASTECTOMY

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RECURRENCE as related to cancer implies the persistence and eventual clinical manifestation of disease not eliminated at the time of treatment. Therefore the recurrence of breast carcinoma in the structures of the chest wall following breast removal indicates that tumor tissue, microscopic in size, was left behind at the time of operation. There has been considerable discussion regarding the influence of wide skin removal on the subsequent development of skin recurrences. However, there appears to be some confusion as to the true etiology of skin recurrences and their significance in relation to treatment and prognosis of carcinoma of the breast. It is with this aspect of the problem that this paper is primarily concerned. Twenty-six cases of carcinoma of the female breast, in which the patients were subjected to radical mastectomy and later developed skin recurrences, form the basis of this presentation.

Since it is well recognized that the problem of breast carcinoma is intimately related to the lymphatic drainage of the breast, it is appropriate to review briefly, at the outset, this somewhat intricate anatomy.

Rouvière describes the cutaneous lymphatics of the mammary region as follows: In the region of the nipple and areola, the lymphatic capillaries form a dense network, the areolar network, which consists of several planes continuous with the superficial cutaneous plexus of the thoracic integument (papillary lymphatic plexus of the skin). From this network arise small lymph vessels which empty into a subdermal plexus, the subareolar lymph plexus. The subareolar plexus receives a large portion of the lymphatics from the breast and gives origin to the great afferent lymph trunks from the mammary gland proper. In the outer circumference of the areola, the subareolar plexus becomes less dense, and its meshes enlarge to form the circumareolar plexus. This plexus merges into the same lymphatic capillary network as the other cutaneous lymphatics draining the anterior thoracic wall and presents the same lymph node connections—that is, the collecting vessels which drain the thoracic cutaneous network converge from the median line and the anteroinferior part of the thorax upon the axilla, penetrate it, and empty into the external mammary group of lymph nodes, chiefly the superior group.

In addition to this principal path of collection, there are three secondary paths of drainage: (1) the supraclavicular lymph nodes; (2) collecting vessels which originate in the median portion of the anterior thoracic wall, accompany the perforating rami of the internal mammary artery, transverse the pectoralis major and intercostal muscles, and finally empty into the internal mammary chain of nodes; and (3) the group of collecting vessels called, by Rouvière, the anterolateral lymphatic path, which cross the midline presternally and run from the integument of one breast to the axilla of the opposite side. This third path was demonstrated by Oelsner. It provides the means by which contralateral axillary nodes may be involved independently of a new primary tumor in the opposite breast.

The cutaneous lymphatic network of the anterior thoracic wall is continuous with the cutaneous network of adjacent regions such as the abdomen, the neck, and the upper extremities. This arrangement, described first by Sappey and then by Handley, was well demonstrated by the classical experiment of Kolodny, who incised the pectoralis major muscle of one side transversely, injected carmine into the caudal portion of the muscle, and noticed that the carmine appeared in the contralateral axillary nodes and in both groups of inguinal lymph nodes.

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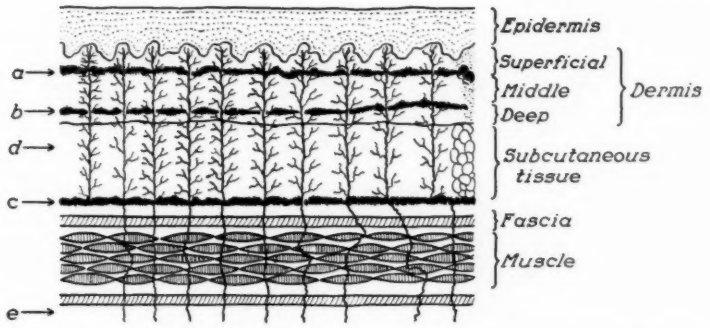

Fig. 1. Diagram showing the anatomical positions of the various plexuses: *a*, superficial cutaneous plexus or Sappey's subpapillary plexus; *b*, deep cutaneous plexus of Arnold; *c*, superficial fascial plexus, the chief path of lymphatic drainage from the breast and surrounding skin; *d*, vertical lymphatic trunks to the dermal papillae; *e*, deep vertical trunks to the underlying structures.

The main plexus into which the cutaneous lymph drains is the so-called superficial fascial plexus. The circumareolar plexus merges with this plexus and is thereby continuous with the cutaneous lymphatics of the entire thorax and adjoining regions. As mentioned previously, continuity is evident from Kolodny's experiment, in which retrograde lymphatic flow was initiated. In the mammary region, the superficial fascial plexus lies in the retromammary and deep subcutaneous tissue just superficial to the fascia of the pectoralis major muscle. Numerous small lymphatic capillaries arising in the papillae of the dermis converge, forming larger vertical vessels which empty into it.

There has been considerable controversy in regard to the superficial cutaneous plexus (Sappey's subpapillary lymphatic plexus). Rouvière indicates that the areolar plexus merges with this network. Handley, on the other hand, was unable to demonstrate a superficial plexus in the dermis and felt that there is a complete independence of adjoining "lymphatic areas" of the skin, which he estimated to be 1/8 to 1/2 inch in diameter. In other words, Handley's conception likened the vertical trunks draining into the fascial plexus to a forest of trees with many branches, none of them connecting with each other.

This anatomical picture is further confused by the frequent description of another lymphatic network, the deep cutaneous plexus, located at the junction of the middle and deep thirds of the dermis. The anatomical positions of these various plexuses are shown diagrammatically in Figure 1. Dermatologic pathologists are likewise divided in their ability to demonstrate either Sappey's or Arnold's cutaneous plexuses, but they are in general agreement that the most important path of drainage from the skin is by the superficial fascial plexus.

While the value of radical mastectomy, established by Halsted and Willy Meyer, is unquestioned in the treatment of carcinoma of the breast, there has been little agreement regarding the method of dealing with the skin. The disagreement seems to be intimately concerned with the various concepts of spread of carcinoma within and beyond the limits of the breast. These concepts are obviously related, in turn, to the respective interpretations of the anatomical considerations of the cutaneous lymphatics in the mammary region.

One of the early workers to study the spread of breast cancer was Handley. His classical work is probably the most often quoted, but at the same time it has received widespread criticism. Handley concluded that the growth of breast cancer to the skin was chiefly by permeation into the lymphatics of the superficial fascial plexus with later extension upward into the vertical skin lymphatics to form clinically visible skin nodules. Heidenhain and Handley showed that permeation of the fascial lymphatic plexus from the primary

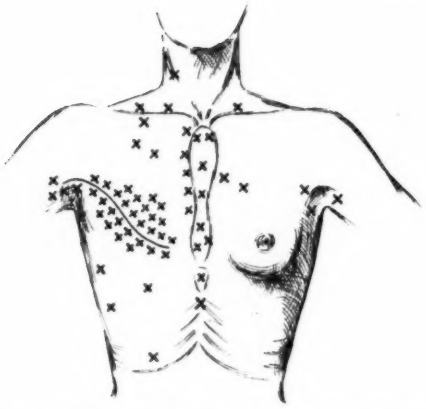

Fig. 2. Diagrammatic illustration of the most common sites of skin nodules. For convenience in diagramming, the right side is assumed to have been the site of primary involvement in all cases.

mammary carcinoma may occur relatively early in the disease and often before the breast is demonstrably fixed to the pectoral fascia. Handley by no means denied the possibility of lymphatic embolic dissemination. In fact, he stated that the axillary and regional lymph nodes are often involved as a result of tumor emboli, thus producing retrograde lymphatic flow. Handley believed that skin nodules appearing over the area where the deep fascia had been excised were simple "efflorescences" without bad prognosis, but that skin recurrences over the intact fascia indicated proliferating growth and certain death. However, as will be indicated later, it would appear that "efflorescent" recurrence is either rare or without appreciably different outlook.

As a result of his studies, Handley practiced and taught relatively narrow skin excision but wide peripheral dissection of the fascia and the subcutaneous level where he assumed the tumor to be permeating. Several workers, notably Cheatle, have taken exception to Handley's permeating theory. As evidence, they have shown that sections cut serially from the infiltrative edge of the primary growth to the regions of metastatic growth will show intervening areas where no permeated lymphatics can be found. This is conclusive evidence, according to these investigators, that spread is by emboli in the lymphatics and not by permeation. Gilchrist and David, in their work on carcinoma of the rectum, further emphasized evidence pointing toward spread by emboli. Handley defended his permeation theory by explaining those intervening areas which were free from permeated lymphatics to be the result of a defensive action of the invaded tissue, resulting in "perilymphatic fibrosis." However, Handley's major contribution has been to emphasize the importance of the superficial fascial plexus as the route by which cancer of the breast gains widespread dissemination to the skin and surrounding regions.

Willis agrees that permeation of breast carcinoma may occur upward from the fascial plexus in the manner described by Handley and may thereby produce skin nodules. However, Willis, as well as Daus and Miescher, places equal importance upon tumor emboli in retrograde lymphatic flow caused by blockage of regional lymph nodes and deep lymphatics. Willis also states that secondary embolic foci may serve as the origin of further permeation. In addition, several references to apparently genuine case reports of hemic dissemination of breast cancer to the skin are given in Willis' book. Batson, likewise, after working with ink-injected material, also emphasizes the fact that venules as well as lymphatics may serve as paths of dissemination to the skin. Willis concludes that skin metastases from carcinoma of the breast do not result from lymphatic permeation, or emboli, or hemic dissemination alone, but are in varying degree the result of all of these routes.

Stiles, writing in 1892 of his findings after using nitric acid fixation of the breast in normal and pathologic conditions, showed a clear understanding of the dissemination of breast cancer. His observations led him to conclude that the "efferent lymphatics of the corpus mammae, of the fat around it, and of the nipple, areola, and skin over it, open either directly or indirectly into the lymphatics of the deep fascia . . . " Stiles denied Sappey's contention that two or three collecting lymphatic trunks leading from the subareolar plexus are the ultimate and only channels for the receipt of lymph from all parts of the breast. He was able to demonstrate clearly small foci of tumor

TABLE I.—CORRELATION OF PREOPERATIVE CLINICAL FINDINGS AND POSTOPERATIVE RECURRENCE OF TUMOR

Case No.	Pregnancy or lactation	Extensive edema	Satellite nodules	Intercostal or parasternal nodules	Edema of arm	Proved supraclavic. metastases	Inflammatory carcinoma	Distant metastases	Axillary lymph nodes more than 2.5 cm.	Ulceration of skin	Edema of less than half of breast	Tumor fixed to chest wall	Fixed axillary nodes	Case No.	Operable?	Type of closure	Follow-up result	Remote metastases?
1	–	–	–	–	–	?	–	–	–	+	–	+	–	1	No	Graft	Dead	Yes
2	–	–	–	–	–	–	–	–	–	–	–	+	–	2	Yes	Primary	Dead	Yes
3	–	–	–	–	–	–	–	–	–	–	+	+	–	3	No	Graft	Dead	Yes
4	–	–	–	–	–	–	–	–	–	–	–	+	–	4	Yes	Primary	Alive	Yes
5	–	–	–	–	–	–	–	+	+	–	+	–	–	5	No	Graft	Dead	Yes
6	–	–	–	–	–	–	–	+	–	–	–	+	–	6	No	Primary	Dead	Yes
7	–	–	–	–	–	–	–	–	–	–	–	+	–	7	Yes	Primary	Dead	Yes
8	–	Physical findings unknown				–		–	Physical findings unknown					8	?	Graft	Dead	Yes
9	–	–	–	–	–	–	–	–	–	–	–	+	–	9	Yes	Graft	Dead	Yes
10	–	–	–	–	–	–	–	–	–	–	–	–	–	10	Yes	Primary	Dead	Yes
11	–	–	–	–	–	?	+	–	–	+	+	+	+	11	No	Graft	Dead	Yes
12	–	–	–	–	–	–	–	–	+	–	–	–	–	12	Yes	Graft	Dead	Yes
13	–	–	–	–	–	–	–	–	+	–	–	+	+	13	No	Graft	Dead	Yes
14	–	–	–	–	–	+	–	–	+	–	–	–	–	14	No	Graft	Dead	Yes
15	–	–	–	–	–	–	–	+	+	+	–	+	–	15	No	Graft	Dead	Yes
16	–	–	–	–	–	–	–	–	–	–	–	–	–	16	Yes	Primary	Dead	Yes
17	–	–	–	–	–	–	–	–	+	–	–	+	+	17	No	Primary	Dead	Yes
18	–	–	–	–	–	–	–	–	+	+	–	+	–	18	No	Graft	Dead	Yes
19	–	–	–	–	–	–	–	–	–	–	–	–	–	19	Yes	Primary	Alive	Yes
20	–	–	–	–	–	–	–	–	–	–	–	–	–	20	Yes	Primary	Dead	Yes
21	–	–	–	–	–	–	–	–	–	+	–	–	–	21	Yes	Primary	Dead	Yes
22	–	–	–	–	–	–	–	–	–	–	–	–	–	22	Yes	Graft Primary	Alive	Yes
23	–	–	–	–	–	–	–	–	–	–	+	–	–	23	Yes	Graft	Alive	Yes
24	–	–	–	–	–	–	–	–	–	–	–	–	–	24	Yes	Primary	Dead	Yes
25	–	–	–	–	–	–	–	–	–	–	–	+	–	25	Yes	Graft	Alive	Yes
26	–	–	–	–	–	–	–	–	–	–	–	–	–	26	Yes	Primary	Alive	Yes

located in the deep lymphatics remote from the primary tumor and "depending for their origin upon the arrest growth of cancerous emboli." Since the diffuse anastomoses of the lymphatic vessels of the fascial plexus between the mammary and surrounding regions were well known to Stiles, it is not surprising that he wrote at this early date of the "importance of removing the retromammary tissue, axillary fat and nodes, pectoral and axillary fascia, along with the breast in all cases of carcinoma." He concluded that it is impossible to predict toward which regional lymph nodes the cancer emboli from any given point of the breast may be conveyed.

It is clear that the lymphatic drainage of the breast is in intimate relation with that of the overlying skin as well as with the skin of the surrounding regions through the rich anastomoses of the subareolar plexus into the circumareolar plexus and finally into the superficial fascial plexus. The superficial fascial plexus is of prime importance in that it serves as the largest afferent lymphatic group from the mammary gland and the skin of the entire body to the regional nodes. Cancer cells from

the primary growth in the breast readily penetrate the lymphatic vessels and may permeate them for varying distances. Emboli from these invasions are broken off and swept to the axillary lymph nodes, grow there, and often completely block the cortical sinuses to further lymph drainage. In consequence, retrograde lymphatic flow may occur, and because the anastomoses of the fascial plexus are so extensive, tumor emboli can be carried to areas remote from the primary tumor, as, for example, to an ipsilateral inguinal lymph node. As Stiles indicated and Kolodny demonstrated, it is impossible to predict how far reaching such a process may be. The emboli may lodge as small foci in the fascial lymphatics, only to permeate later into the vertical skin lymphatic capillaries, or they may be swept directly into the vertical skin vessels.

It would appear, therefore, that much of the recurrence of tumor in the skin originates from malignant cells present in the fascial plexus, and that these malignant cells are capable of spreading to surrounding anatomical regions. Undoubtedly some skin nodules are the result of direct permeation of the deep lymphatics and vertical spread into the cutis without the aid of embolization. Hemic dissemination probably accounts for a small percentage of skin recurrences. All of these methods of spread would appear to be operative in the production of local recurrences. To attempt to define closely the exact route in each instance is of academic interest only. The important thing to be borne in mind is that the superficial fascial plexus serves as the primary route of lymph drainage from the skin of the mammary region as well as the breast itself, and because of the extensive anastomotic potentialities of this plexus, combined with retrograde lymphatic flow, skin recurrence by any route necessarily implies true metastases.

Another factor concerns the involvement of axillary lymph nodes by tumor. As has been indicated, extensive lymph node involvement is often followed by retrograde lymphatic flow. Since the incidence of axillary metastases is extremely high in patients developing skin recurrence, it seems probable that retrograde embolization is frequently responsible for the skin recurrence.

Although there are several instances in the literature in which excision of skin recurrences near the operative scar was followed by freedom from further disease for as long as 10 to 15 years, such instances are extremely uncommon and suggest the occasional inoculation of tumor cells at the time of operation. Unusual happenings of this nature probably contribute little to an understanding of the real problem.

Lewis and Rienhoff, in reporting the results of radical mastectomy for carcinoma of the breast at Johns Hopkins Hospital from 1894 to 1931, consider that local recurrences represent either inoculations at the time of operation or uninterrupted tumor in the cutis that was transected at operation. They have substituted the term "regional metastases" for "regional recurrences," though this seems paradoxical considering their own interpretation of skin recurrence. The outcome of their studies, in line with the principles laid down by Halsted, has been the technique of wide skin excision followed by a Thiersch graft over the operative defect. Lewis and Rienhoff conclude in their paper that "regardless of the extensiveness of the disease, so long as it remains confined within possible operative limits, it is incumbent on the surgeon to perform the most radical and meticulous operative procedure, including a far wider exsection of skin than has been customary in even the Halsted Thiersch graft procedure as performed in the majority of instances in the past, for it is only by this method that the proper amount of skin and subcutaneous tissue will be sacrificed. There alone exists the possibility of reducing local recurrences to a minimum."

Another school of thought led by Auchincloss agrees with the Halsted principles but believes that they do not go far enough, as the spread of cancer peripheral to the excised skin takes place in the tissue immediately beneath the cutis. Therefore, a more careful and painstaking dissection of the remaining skin around the excision must be made so as to leave no fat attached to the undersurface of the skin.

White and his co-workers have renewed interest in the work of Handley. White considers, as do most surgeons, that a primary closure is preferable to skin graft if it does the patient no harm. He states that a minimum

width of 15 centimeters of skin should be removed, and that by liberal undercutting the flaps can usually be easily approximated without undue tension. He believes that the advocates of skin grafting have adopted an empirical attitude that is not yet supported in fact.

Though it has been rather widely implied that the incidence of postmastectomy skin recurrence is in general a reflection of the operative technique, little has been done in retrospective study of other underlying factors which might influence the later development of skin recurrence, or in consideration of whether by means of excessively wide skin removal any material gain is accomplished in increasing the long term survival of patients.

Out of 450 cases of carcinoma of the breast from the records of the Ellis Fischel State Cancer Hospital we found 26 cases of biopsy-proved skin recurrence developing in or adjacent to the operative field in patients that had had radical mastectomies. It is on these 26 cases that our discussion is based. No attempt has been made to calculate percentage recurrence since in many of the 450 cases sufficient time has not elapsed for the appearance of future inevitable skin recurrences.

The average incidence of local recurrence following radical mastectomy can be obtained from a general review of the literature. Lewis and Rienhoff report a rate of 26.6 per cent in 541 patients who had careful follow-up attention. In White's series of 254 patients, 23.5 per cent developed local recurrence. Hoopes and McGraw report a 15 per cent local recurrence in 246 patients. Rodman reports the extremely low rate of 2.2 per cent local recurrence in 132 patients, of whom 61 per cent survived for at least 5 years. It should be noted, however, that these patients were personally selected. Other contemporary writers generally report figures of 10 to 30 per cent local recurrence. It is interesting to note that prior to the initiation of the radical mastectomy operation for carcinoma of the breast, surgical masters like Billroth, Czerny, Fisher, and Gussenbauer reported local recurrences in 62 to 82 per cent of their patients.

In reviewing our cases we have attempted to determine what correlation exists between certain preoperatively recognized clinical findings known to influence adversely the prognosis of breast carcinoma and the subsequent manifestation of recurrences. Table I shows the clinical findings and the operative sequelae.

In 4 cases (Nos. 5, 6, 14, 15) operation may be considered to have been hopeless. These patients presented initially suspected, and later obvious, distant metastases. Operation was performed in order that the patients might receive the benefit of the existing doubt in regard to the suspected metastatic disease. If one accepts the summation of local factors constituting inoperability as outlined by Stout and Haagensen, in 6 additional patients (Nos. 1, 3, 11, 13, 17, 18) the cancer might have been considered inoperable, bringing the total to 10. All of these patients have now died of generalized disease, pulmonary metastases being present in 8 of the 10. Of the remaining 16 patients, 8 presented at least one highly unfavorable clinical sign. Ten of these 16 are dead of carcinomatosis, and all of the remaining 6 patients still living have shown remote disease and may be expected to succumb to the cancer in due course of time.

It has been repeatedly shown in published reports that the incidence of local recurrence is greatly increased when axillary metastases are present (16, 17, 18, 19, 20). Warren and Tompkins have produced some interesting data on this point. They have shown that skin recurrences are three times more common when axillary nodes are invaded by tumor, and that the incidence of skin recurrence increases in direct proportion to the percentage of lymph nodes involved by disease. In their series there was only a 7 per cent skin recurrence when no axillary lymph nodes were involved, but a 21 per cent skin recurrence when all of the axillary nodes were invaded. They maintain that it is reasonable to suppose that patients in whom metastases have occurred to the axillary lymph nodes should also have other metastases to the skin over the chest wall, thus increasing the likelihood of recurrence in the scar. They interpret their data to suggest that local recurrences are of the nature of metastases rather than implants. In emphasis of this interpretation, Warren and Tompkins note that the patients in their series who had no axillary lymph node involvement

TABLE II

No. patients	Total nodes involved	No. nodes dissected	
	Per cent	Average	Range
2	0	17	4–30
9	less than 25	20	8–40
1	25–50	15	
6	50–75	14	9–25
4	75–99	20	7–41
4	100	12	10–16

TABLE III.—TIME AVERAGES

(1) From operation to appearance of nodules... 14.4 mos.
  (a) Skin graft cases... 13.9 mos.
  (b) Primary closure cases... 16.5 mos.
(2) From operation to death (20 patients)... 24.3 mos.
(3) From operation to time of writing, for those alive (6 patients)... 35.5 mos.
(4) From appearance of skin nodules to death... 10.8 mos.
(5) From appearance of skin nodules to time of writing... 22.1 mos.
(6) Average age
  (a) All 26 patients... 51.7 years
  (b) Patients living (6)... 55.7 years
  (c) Patients dead (20)... 50.6 years

but who developed skin recurrence (a total of 81) died of widespread metastatic cancer.

Table II gives, in rough figures, the percentage of axillary lymph nodes found to contain cancer, in our patients. Only 2 of the 26 patients were free from apparent metastases to the axillary lymph nodes, as determined by examination of the operative specimen. In 1 of these patients, only 4 lymph nodes were examined, so that it cannot be definitely stated that there were no metastases. Only 2 of the 15 patients in whom 25 per cent or more of the lymph nodes were involved are still living. On the other hand, 4 of 11 patients with less than 25 per cent of the axillary nodes involved are still living, though all have widespread disease.

In general our policy has been to excise as much skin as is compatible with primary closure except where other considerations such as a small breast, a large tumor, or a tumor superficial in the breast or involving the skin are present. Fourteen of our patients required skin grafting, and 12 had flap closure of the wound. It is not our purpose to contribute to the controversy regarding what constitutes adequate skin removal. However, it is difficult to imagine what skin grafting contributed to the overall prognosis in the 14 patients in whom it was carried out, or what flap closure contributed in the other 12.

The location of the skin nodules is shown diagrammatically in Figure 2. It is seen that the most common site of recurrence is along the margin of the scar or at the periphery of the grafted area. One would expect this site to be the most frequent on the basis of simple mechanical dynamics. Handley explained the fact that recurrences are found most frequently near the site of the primary tumor on the basis of a centrifugal spread of cancer. The next most frequent site is over and parallel to the sternum. The collecting lymphatic vessels which accompany the perforating branches of the internal mammary artery undoubtedly account for most of the recurrences along the sternum. These lymphatic vessels are in direct communication with the superficial fascial lymphatic plexus. Less often, involved areas are found in and near the axilla and supraclavicular regions of the same side. Several of our patients developed skin nodules in or around the axilla of the opposite side.

The averages of various time intervals relative to our series are listed in Table III.

CONCLUSIONS

While radical mastectomy has been universally adopted as the most effective method of dealing with carcinoma of the breast, the reported incidence of skin recurrence following this procedure remains at between 10 and 30 per cent. Surgeons, in an attempt to lower this incidence, have proposed many technical maneuvers and variations in procedure, usually aimed at increasingly wide skin removal. However, it appears that such technical attempts at elimination of local recurrence are of little value in effecting a cure of carcinoma of the breast. Our study of the 26 patients at the Ellis Fischel Hospital who developed skin recurrences following radical mastectomy clearly shows that the tumors present in these cases were advanced and aggressive. Indeed, in 10 of the 26 patients there was evidence of such advanced disease that operation might have

been considered futile by generally accepted clinical criteria of operability.

Therefore, it would appear to be a proper concept that skin recurrences indicate that not all of the carcinoma was accessible to the surgeon at operation, regardless of how radical his procedure, and that the cancer was inoperable.

This concept is based upon the fact that the route of metastasis of breast carcinoma to the skin is usually by the superficial fascial lymphatic plexus with vertical growth into the skin. Permeation, as proposed by Handley, and embolization in retrograde lymphatic flow are probably the chief modes of metastasis, the latter appearing to be the one of primary importance. When the axillary lymph nodes are uninvolved and yet skin metastases appear later, it may be assumed that hemic embolization, direct lymphatic permeation, or independent lymphatic embolization without retrograde flow was the manner of dissemination (20). By any of these means it may be anticipated that remote metastases may also develop. Although there can be little question that recurrences may also result from direct implant of tumor cells, this would seem to happen quite rarely and in all likelihood is usually associated with wide lymphatic involvement as well.

The safe amount of skin to be removed in radical mastectomies has given rise to great disagreement. Bearing in mind the lymphatic system of the breast and surrounding integument, incision of the skin at a distance beyond the tumor equal to the distance permitted between the tumor and the chest wall should provide more than ample skin removal. Wide skin excision, although it might conceivably diminish the clinical manifestation of skin recurrences, would not, in our opinion, improve the 5 year survival of patients. This conclusion is borne out by careful comparison of the reports of those who regularly practice skin grafting and those who do not. If lymphatics of the superficial fascial plexus have become involved, either by embolic arrest or permeation, wide skin removal does not prevent recurrence.

It seems reasonable to conclude that skin recurrence is but the indicator of the recrudescence of more widespread disease already clinically apparent or soon to become so. It is our opinion that treatment of the recurrence, either surgical or radiotherapeutic, adds little or nothing to patient's life expectancy.

Thus, decreasing the incidence of skin recurrence would appear to be dependent among good surgeons not on the commonly discussed variation in surgical technique but on the earlier diagnosis and surgical treatment of breast cancer and on the adherence to even narrower criteria of operability than have been previously followed. In fact, it is believed by some workers (19) that patients with advanced but still technically resectable carcinomas live less long after operation than do those who are refused radical mastectomy.

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EDITORIAL

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SEPTEMBER, 1947

THE IMMEDIATE CARE OF NERVE AND TENDON INJURIES

FEW types of injury can impose a greater handicap upon the working man or the boy or girl beginning the active years of life than a deep laceration of the palm or forearm with division of nerves and tendons. It is in these two groups, in particular, that such injuries are most likely to occur. The workman handling metal plates and strips, working with glass sheets or bottles or with heavy machinery with shearing edges; the youngster at play, falling with a bottle or tumbler clasped tightly in his hand or pushing his forearm through a glass door or window; the adventurous schoolboy rummaging in fascinating heaps of discarded tin cans, automobile fenders and glass containers; and the luckless individual of any age involved in an automobile accident—any one of them can confront the surgeon, and at any hour, with a severe injury that taxes both his surgical judgment and his surgical skill.

As with every surgical procedure an accurate preoperative diagnosis enables the surgeon to work effectively and with assurance. No one emphasized this fact more clearly in his writings and teachings than the late Torr Harmer of Boston, or described more clearly how with a few simple tests of movement and of sensory function, and usually without removing the first aid dressing, the surgeon could recognize with certainty the division of nerves and tendons and identify which were injured and which intact.

When one is suddenly confronted with a deep wound in which the tissues are uniformly discolored with blood and in which all the anatomical structures seem to be fused into a single soft mass of dusky red, areolar tissue and blood clot, one needs both the courage and assurance that come from an exact recognition of the extent of the injury.

The prompt healing and the freedom from postoperative wound reaction and discomfort that follow operative procedures when no hematoma formation or wound infection has taken place are nowhere more essential for a successful outcome than in the case of nerve and tendon injuries. A divided nerve must heal with a minimum of fibrous tissue formation if nerve axons are to find their way into the distal segment of the divided nerve. Tendons must again glide back and forth if function is to be restored. The induration and fibrosis that follow extravasation of blood into the soft tissues and that are so pronounced in tissues in which infection has taken place can cause compression of injured nerves that results in interruption of nerve impulses, scar

formation at the site of union of divided nerves that blocks downgrowth of nerve axons, and fixation of sutured tendons that makes free gliding movement difficult or impossible. The fibrous tissue formation that could conceivably strengthen the abdominal wall after closure of a hernial opening or reinforce the site of closure of a perforated bowel can make the restoration of function after the repair of nerve and tendon injuries exceedingly difficult or even impossible to accomplish.

How can one be assured of the maximum chance for success in the repair of such an injury? A number of factors must be considered, but first in importance is the *time that has elapsed*. One could almost assert categorically that if the injured patient is seen promptly—within a half hour after injury has occurred, and if the first aid treatment has been limited to the immediate application of a sterile bandage and a compression dressing it should be possible to convert any lacerated wound without extensive tissue loss into a clean surgical wound that can be repaired and that can be expected to heal by primary union. This possibility diminishes definitely with every half hour that elapses, and after three hours one would be unwilling to attempt primary repair of divided nerves and flexor tendons. Lost blood can be replaced by transfusion; there is no compensating substitute for lost time.

Other factors in addition to the time that has elapsed should be thoughtfully considered. The possibility that bacterial contamination has been added to an open wound as a result of overzealous first aid treatment must be kept in mind. More and more, surgeons in industry and men who constantly see patients with compound wounds emphasize the importance of covering the open wound with a sterile dressing at the earliest moment, applying a compression dressing and immobilizing splint, and avoiding any examination of the wound, any chemical applications, or any effort to tie off bleeding vessels as a first aid measure. Simple compression with an elastic bandage or direct manual pressure over a sterile dressing can arrest even arterial bleeding. Rarely is a tourniquet required in first aid treatment; too often it accelerates bleeding instead of arresting it. The late Mont Reid often commented on the frequency with which bleeding from an open wound ceased when the tourniquet was removed and the part elevated.

The circumstances under which the injury was sustained deserve consideration. Clean cut wounds of clean hands, caused by sharp metal or glass, and sustained indoors are least likely to be contaminated. At the opposite end of the scale are wounds from more blunt objects sustained out-of-doors, perhaps in a dirty street or in an accident on the farm. Hands and forearms covered with grease and oil are not necessarily infected; one often sees wounds of greasy hands heal kindly even though the grease has thoroughly stained the exposed tissues of the open wound.

It is hardly necessary to add that a well equipped operating room with adequate assistance from anesthetist and house staff is essential in carrying out efficiently what can be a trying and tedious surgical procedure. To attempt to repair divided nerves and tendons in unfavorable surroundings and without sufficient aid almost certainly results in failure.

The difficult emergency operation often imposes a psychological handicap on the surgeon and his assistants. It comes unexpectedly. It thrusts itself like an unwelcome visitor into a well ordered routine. Often it is difficult to postpone planned procedures and give one's undivided attention to the unanticipated problem in which "time is of the essence." Here again necessity knows no choice, and the

best interests of the patient will be served if the surgeon can promptly and whole heartedly transfer his attention to the new and unexpected task.

In the operating room the preparation of the wound, transformation of the contaminated wound into a clean surgical wound, is one of the most important steps in the surgical treatment. Zinninger, Hermann, Altemeier, and their associates at the Cincinnati General Hospital have performed a splendid service for the medical profession in their excellent motion picture demonstration of how wound cleansing and preparation for repair can and should be carried out. Their demonstration has been seconded and emphasized by an excellent film on the surgical preparation of patients with compound injuries prepared by the Medical Department of the Navy. Insistence on patience, thoroughness, and avoidance of mechanical and chemical trauma emphasizes surgical principles which are everywhere accepted, and which these films illustrate so graphically. If they continue to receive the publicity and attention they deserve the standards of care will be measurably improved wherever surgical work is being done.

When wound cleansing is complete the surgeon must make the final decision as to whether immediate repair should be undertaken. If he is satisfied that the open wound has been transformed into a clean surgical wound, and if it can be closed without tension there is every logical reason for immediate suture of divided nerves and tendons. If he is still in doubt it is wise to close the wound without tension and postpone the repair until healing has taken place and inflammatory reaction subsided. Repair and incomplete closure are inconsistent with one another and fail to give satisfactory results. The local use of chemotherapeutic agents favors hemorrhagic exudation and hinders rather than helps the healing process. Surgeons for the most part agree that sulfonamides and penicillin are of very great value in preventing spreading infection, but they cannot safeguard the patient if surgical principles are neglected or ignored. Too many failures in the past have demonstrated that one cannot safely extend the indications for repair and closure of compound injuries because improved chemotherapeutic agents are now available. They constitute an added safeguard but not a substitute for time tested methods.

In the operative procedure two helpful aids are available to simplify the operation. The use of a bloodless field secured with the aid of a blood pressure cuff is an important advantage always available to the surgeon operating upon the extremities. Extension of the wound of injury, both above and below, by incisions that do not cut across flexion creases permits adequate exposure and enables one to identify injured structures with certainty, something which is exceedingly difficult to do if one begins the dissection in the uniformly discolored tissue present at the level of the injury. Gentleness in handling tissues, the use of fine nonabsorbable suture material, accurate apposition of divided tendons and nerves, complete hemostasis, protection of the suture line of the divided structures with soft areolar tissue, and careful closure of the fascial envelope that surrounds the structures of the forearm, that holds them in place and separates them from overlying subcutaneous tissue and skin —all are simple principles which are accepted by good surgeons everywhere, and all are of great importance in achieving a satisfactory result.

The younger surgeon whose experience has been largely with military casualties, and whose surgical decisions have been guided by directives laid down as broad policies, may be perplexed at times in transferring his war ex-

periences to the problems of civilian surgery. The opportunity to see the patient promptly after injury, to care for him immediately, to give him as much time as is necessary for meticulous surgical care and to follow him day by day all help to make possible procedures that could not be attempted with safety under the conditions which obtain in military surgery. The surgical skill that has been acquired in handling large numbers of cases can be of inestimable value if it is combined with a realization of the opportunities associated with individualization rather than "mass treatment" of surgical problems.

Alertness, good surgical judgment, and the willingness to undertake a difficult surgical task promptly, rather than to "see the patient in the morning," will secure the optimum results for patients with serious nerve and tendon injuries. When one recalls how completely an individual's outlook on life can be changed by such an injury, whether the patient is a workman whose livelihood depends on the function of his hand or a youngster with life stretching before him, it is not too much to ask that he shall receive the most skillful aid that good surgery can bring to him.

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CANCER AND POLITICS

The Operation on Grover Cleveland

M. G. SEELIG, M.D., F.A.C.S., St. Louis, Missouri

EVERY story is set off by its accompanying background. Sometimes this scenery has to be created by literary artifice; in other instances, nature or circumstance supplies it. In 1893 one of the most dramatic surgical operations in American history lent color to a period that was packed with sufficient explosive tinder to alarm the best and most patriotic minds of our people. The patient was Grover Cleveland, the President of the United States. The operation was the removal of his left upper jaw, which was the seat of malignant disease.

Age, overwork, obesity, the attritional effect of his years in office with their influence on prognosis are not insignificant details of background; but, after all, they constitute technical data of purely surgical significance. At the very heart of the concern of those charged with the destiny of our country in 1893, was the fact that in one pan of the balance lay the life of a man, and in the other all the elements of an incipient political revolution.

Here is the picture: In November of 1892, Grover Cleveland won his second election for the Presidency of the United States over his opponent, Benjamin Harrison and on March 4, 1893, took office. This was a period of exciting unrest, due to the heavy exportation of gold by the United States; the suspension of free coinage of silver by India; the lack of confidence in public credit, due to the shortage of gold in the treasury of the United States; the fear of a silver basis; the unwise speculation of this era and the problem of general unemployment. These various factors precipitated on June 27, 1893, a devastatingly disastrous panic. One of the first acts of President Cleveland was to call a special session of Congress for August 7, 1893, to repeal, absolutely and uncompromisingly, the Sherman Act. The essential goad for driving Congress over partisan hurdles, on to this repeal, was that very "defiant frankness" and "blazing indiscretion" which were in reality Cleveland's powerful virtues even though construed by his foes as his faults. It was, literally, on the very eve of this battle that the President developed sarcoma of the left superior maxilla.

On June 18, 1893, just 50 days before Congress was to meet in special session, Dr. R. M. O'Reilly, who later became Surgeon General of the United States Army, examined the President and discovered an ulcer of the left side of the hard palate; this lesion was about one and one-half centimeters in diameter, encroaching on the soft palate and the underlying bone. A portion of the lesion was excised for biopsy and was pronounced by the pathologist of the Army Medical Museum (who did not know the name of the patient) to be highly suggestive of malignancy. Dr. Joseph D. Bryant, an intimate friend of the President and a New York surgeon of renown, who was called in at the suggestion of Dr. O'Reilly, advised an immediate operation. The President accepted this counsel without qualification.

From what has already been said regarding the gravity of the economic unrest in the country, it will be appreciated readily that the diseased area in the mouth of President Cleveland, though no larger than a silver quarter, loomed on the horizon in ecliptic proportions. The challenge of surgery had been accepted by the patient; but had the public got word of what was brewing, the consequences would have been unpredictable. Secrecy was therefore the order of the day; and as we shall see later, every circumstance connected with the incident was shrouded from both the members of Congress and the public at large.

Unfortunately, however, one newspaper man, under the by-line of "Holland" (E. J. Edwards) published, in the *Philadelphia Press* of August 29, 1893, a report that the President had undergone a serious operation for the removal of some teeth

From the Department of Research of The Barnard Free Skin and Cancer Hospital, St. Louis, Missouri.

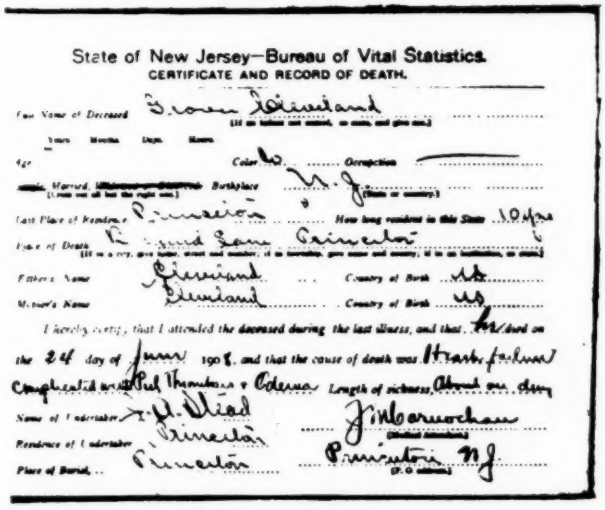
and a good deal of the bone from the upper jaw. Many of the details set down by "Holland" were uncannily correct. But the fact that the President had opened the special session of Congress on August 7th, presenting no evidence of scar and in perfect voice, tended to discount the report of Holland as more or less of a canard or at best as a gross exaggeration. The Editor of the *Philadelphia Public Ledger*, Mr. L. Clarke Davis, a personal friend of Mr. Cleveland, characterized Holland's report as an infamous exploitation of a toothache and a cancer fake. Other newspapers denied that any operation had been performed. And so, from the date of operation, July 1, 1893, until Dr. W. W. Keen published an authentic, play by play account of the incident, 24 years later, and 9 years after Grover Cleveland's death, the general public was kept in ignorance of all the facts in the case.

Before outlining the information detailed in Dr. Keen's account of the operation, the postoperative course, and the measures taken to preserve secrecy, it may not be amiss to call attention to the fact that of our 32 Presidents, two have been subjects of malignant disease—Grant and Cleveland. This represents a proportion of approximately one to sixteen, a figure that is strikingly lower than the one in eight that is generally presumed to be the actuarial ratio of cancer incidence. All such figures should be taken with more than a grain of salt, as anyone will realize who patiently analyzes the most authentic historic accounts of the lives and deaths of our Presidents, only to discover the difficulties in the way of establishing accurate diagnoses either of the illnesses they endured or the specific causes of death in any but a negligibly small number of them. There is no more warrant for concluding that the Presidency of the United States carries with it a partial immunity to cancer than there would be justification in assuming that the American electorate suffers from a presidential assassination complex, because in the comparatively short period of 81 years, from 1865 to 1946, we have murdered over 18 per cent of our Chief Executives.

As further evidence of the elusive value of statistics, is the fact that, of the Presidents who were subjects of cancer, 50 per cent recovered. Cleveland was operated upon in 1893, and died fifteen years later of cardio-vascular-renal disease, uncomplicated by anything that resembled either recurrence of the original tumor or metastasis springing from it. His various biographers refer to recurrent attacks of gastrointestinal disease, but one finds nothing indicating that these attacks were, in any way, associated with gastrointestinal malignant disease—primary or metastatic. A semi-official public statement, signed by his friends, Dr. Joseph D. Bryant, Dr. George R. Lockwood, and Dr. J. M. Carnochan, and published the day of his death, stated that "heart failure, complicated with pulmonary thrombosis and oedema, were the immediate causes of his death." This statement also reiterates the facts that "Mr. Cleveland for many years had suffered from repeated attacks of gastrointestinal disease. Also that he had long standing disease of the heart and kidneys." Despite these seemingly accurate data, a copy of the official death certificate, kindly furnished me by the Department of Vital Statistics of the State of New Jersey, records the duration of the President's illness as "about one day." A photostatic copy of this death certificate is herewith reproduced.

The final cause of the death of President Cleveland is significant, chiefly in its bearing on the dramatic incident of the operation for the removal of the President's left upper jaw. The preexisting cardio-vascular-renal disease must have added greatly to the concern of the surgeons. The entire episode created an explosive touch-and-go situation that not only called for the secret restoration of the health of the President, but also demanded every ounce of precaution to avoid serious postoperative complications, and to assure the resumption of his activities by the President within the short period of about six weeks before Congress met in special session. These ends must be accomplished by surgeons working on a subject who was, on any account, a poor surgical risk. It is true that the famous New York internist, Dr. E. G. Janeway, after a careful physical examination, issued a clean bill of health, reporting normal lungs, good pulse, little if any arteriosclerosis, and almost normal kidneys. But Dr. Keen, in discussing the hazards of anesthesia, remarks that much anxiety was due to the fact that the patient was 56 years old, very corpulent with a short thick neck, in general representing the apoplectic habitus, and harassed almost to the point of physical and nervous exhaustion by the cares of office and in particular by the unrelenting pressure of patronage and office seekers.

These details, together with those set down earlier concerning the unsettled social, economic, and political state of the Nation, furnish us all that is necessary as a background for the fateful operation, which we shall now describe. All the details embraced in this particular part of the recountal rest, by necessity, on one source, namely, the story published by Dr. W. W. Keen in the *Saturday Evening Post* on the 22nd of September, 1917 (page 24). Dr. Keen, a Philadelphian, who was 80



State of New Jersey—Bureau of Vital Statistics.
CERTIFICATE AND RECORD OF DEATH.

Grover Cleveland

Princeton

Princeton

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Death certificate.

years old when he wrote this article, had enjoyed international pre-eminence as a surgeon for many decades. He was, therefore, the logical man to serve as Dr. Joseph D. Bryant's consultant. Dr. Bryant had as aids and additional consultants, Dr. Janeway, the internist, Dr. Ferdinand Hasbrouck, the dentist and nitrous oxide gas anesthetist, Dr. R. M. O'Reilly, ether anesthetist, and Dr. John Erdmann, who was Dr. Bryant's assistant in private practice.

Since it seemed that secrecy could be best preserved by having the operation performed on a boat, it was decided to make use of the yacht "Oneida" whose owner Mr. E. C. Benedict, of New York, was a devoted and intimate friend of President Cleveland. At different times of the day and night of June 30, 1893, carefully avoiding all scrutiny, Doctors Janeway, O'Reilly, Hasbrouck, Erdmann, and Keen, boarded the yacht, lying in New York harbor. That same night, the President, with his secretary of war, Daniel Lamont, and Dr. Bryant, boarded the yacht, which had been converted, temporarily, into an efficient hospital ship. Mr. Benedict was also a member of the party.

On the morning of July 1, the yacht proceeded up the East River at half speed, while the operation was performed. Under nitrous oxide gas anesthesia, Dr. Hasbrouck extracted the two upper left bicuspid teeth. Then, after Doctor Bryant had made the necessary incisions in the roof of the mouth, ether was substituted for nitrous oxide gas during the rest of the operation, throughout which Doctors Keen and Erdmann assisted Doctor Bryant. The entire left upper jaw was removed from the first bicuspid tooth to just beyond the last molar tooth. A small portion of the soft palate

was removed, but the orbital plate was left intact. The antrum was found to be partly filled by a gelatinous mass, grossly sarcomatous in nature and confirmed later to be sarcoma by Dr. William H. Welch, the Professor of Pathology at Johns Hopkins University. The entire operation was done within the mouth, thus avoiding an external scar as visible evidence of the performance of an operation. The skillful use of pressure packs, hot water and the galvanocautery made it possible to complete the operation with the aid of only one ligature. During the course of the operation, which lasted approximately one hour, about six ounces of blood was lost. At the completion of the operation, the wound cavity was packed with gauze and the patient was returned to his bed in excellent condition. The postoperative course was uneventful, the patient's temperature never rising higher than 100 degrees F. and his pulse averaging 90. Unfortunately, however, speech, "labored but intelligible," while the wound was packed, was wholly unintelligible when the packing was removed, "resembling the worst possible case of cleft palate." This defect was corrected by the New York dentist, Dr. Kasson C. Gibson, who fitted the President with an artificial jaw of vulcanized rubber.

The second day after operation, the President was out of bed, and two days after that, the evening of July 5th, the yacht sailed up Buzzards Bay, where the patient was transferred by launch to Gray Gables. He walked to his residence with apparent ease. A few days after this, Dr. Bryant, suspicious that a remnant of tumor tissue had escaped removal, requested Doctors Keen, Erdmann, and Janeway, again to board the Benedict yacht "Oneida," where, on July 17th, under the same secret precautions as previously, all suspicious tissue was excised and the entire surface seared with the galvanocautery. Again the President was up and about on the second postoperative day.

On August 5, 1893, just twenty days after the second operation, President Cleveland arrived in Washington, D. C. in order to direct the strategy of the repeal of the Sherman Law. The law was repealed. It is generally conceded by authorities in the field of history, that, had Cleveland's opponents suspected the true state of affairs concerning his physical compromise, the gravity of the consequences would have been unpredictable. Doctor Keen, in his account of the operation, remarks that if knowledge of the performance of the operation had leaked out, at the time, we can "only surmise and shudder" over what the national and political consequences might have been.

Grover Cleveland died fifteen years, lacking a month, after the removal of his jaw. One finds nowhere in literature any credible, specific statement regarding the cause of death. As has already been noted, the death certificate, is not satisfactory. There circulated a rumor, a short time after Mr. Cleveland died, that his death was due to abdominal cancer. It is impossible to discover whether or not this is true; or, if true, whether the lesion was an independent primary growth or a metastatic lesion. None of the full length biographies clears up the question. Confronted with this dilemma, even at this late day, I inquired of Mr. Cleveland's widow, Mrs. Frances Folsom Cleveland Preston, who wrote me saying that she regretted her lack of information concerning "the doctors' side of it all." She goes on to say further in her letter that, "I never knew what it was, but it must have been the same deadly enemy, internally. The doctors have all gone. No one really knows. I was close to the suffering and distress of mind and body and could be thankful when the relief came."

One participant in the dramatic incident, Dr. John F. Erdmann, who assisted Dr. Bryant, is still living, in the city of New York, active in the practice of surgery. I wrote him for information, and he has been kind enough to tell me that he knows that Mr. Cleveland died of intestinal obstruction but does not know whether or not the obstruction was due to a malignant growth. Dr. Erdmann feels that if the obstruction were due to a malignant growth, it must have been a second primary tumor, because it developed almost fifteen years after the removal of the jaw tumor. Dr. Erdmann further states that he had been told by Dr. Bryant that Dr. Keen was called into service as consultant in order to "assume responsibility, in part, in the event of a fatality."

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REVIEWS OF NEW BOOKS

THE second edition of Handfield-Jones' *Surgery of the Hand*[1] belongs in the library of every man who handles infections or injuries of the hand. It has been developed from a long experience in lecturing on the subject and from the need for expressing clearly and tersely the salient features of hand surgery. The author has been able to set down in minimum space the maximum of information. His approach is anatomic and physiologic; each condition is considered first from the standpoint of the structure of the area concerned, from which are derived the directives for therapy. The functional significance of the hand and the various features thereof are constantly kept in the foreground and stressed. The resident and young surgeon entering his surgical practice can receive no better theme for care of wounds and infections of the hand than is given in this book.

With the tendency nowadays to rely on chemotherapeutic and antibiotic agents to compensate for errors in surgical care the properly sobering influence of Handfield-Jones' book in emphasizing principles is thoroughly in order. One cannot express this better than the author who states,

"Although in penicillin we have placed in our hands a weapon of great power, let us remember it does not diminish one whit the need of early diagnosis and exact treatment if we are to achieve our real object."

As evidence of the soundness of his teaching it is observed that the section on infections has been increased by five pages by the inclusion of material on the sulfonamides and penicillin. Chapters have been added on amputations and burns, both somewhat stimulated by recent war experiences.

One might differ from the author in some things, particularly in the problem of early resumption of motion in injured and infected hands, and in some phases of burn therapy. These differences hinge largely on differing concepts of the significance of rest for injured and inflamed tissues, and entirely on the period during which rest should be enforced. On the question of primary tendon repair the reviewer differs somewhat on the indications for primary repair and on the feasibility of repair of tendons within their sheaths. The time limit of 8 hours within which tendon suture may be safe seems a bit too long. Four hours has seemed a safer limit. Under favorable conditions it is possible by proper enlarging incisions to expose and repair nerves and tendons as a primary operation. Repair within the digital sheaths has been shown to be feasible provided it is accomplished within 2 hours after injury, provided certain prerequisites obtain and a definite technique is followed.

These criticisms do not detract from the value and significance of this book and the soundness of its teachings. If followed by every house officer and every general surgeon the level of care of wounds and infections of the hand would be immeasurably raised.

MICHAEL L. MASON.

A COMPREHENSIVE survey by Dr. Harold Thomas Hyman of the fields of general medical practice is compressed into four volumes, each of about 1000 pages.[2] These volumes are serviced by a special, separately bound index. The work, intended as a complete text for the general practitioner, is divided into five sections: bodily injuries and responses to injury; disturbances of the systems of communication and co-ordination; disturbances of end organ systems; techniques of diagnosis and therapy; and prognosis. The basic material, that of internal medicine, is very well presented; among other fields covered are convalescence, dentistry, dermatology, emergency surgery, neurology, obstetrics, and pediatrics. Incorporated is contributory information furnished by such preclinical sciences as anatomy, bacteriology, pathology, physiology, and serology. The subjects of physical diagnosis, laboratory methods, clinical pathology, electrocardiography, pharmacology, and therapeutics are treated. Illustrations are profuse.

To assist in the interpretation of symptoms 319 "tables of differential diagnosis by presenting symptoms and signs" are included. Integration of the text is accomplished by means of an index which provides references to the separate presentations and to the tables, and by means of cross references. It is, therefore, usually possible, after taking a history and examining the patient, and after reference to the proper table, either to make a definite diagnosis or to discover what further procedures are necessary for diagnosis and, thereafter, to outline a program for the relief or cure of the symptoms which led the patient to seek medical advice.

This ambitious project is an encyclopedia for the general practitioner, intended for reference as regards the patient rather than for straight reading. Although it is questionable whether these volumes make superfluous the use of other texts of more limited scope and doubtful whether the aim set forth can ever be completely accomplished, it must be admitted that the work is astonishingly successful. The general practitioner cannot fail to derive benefit and obtain help in his work from its use and study.

WALTER H. NADLER.

IT is impossible in a few paragraphs thoroughly to review the monograph entitled *Further Studies in Encephalography*.[3] While of modest size, (only 104

[1] SURGERY OF THE HAND. By R. M. Handfield-Jones, M.C., M.S., F.R.C.S. A William Wood Book. Baltimore: Williams & Wilkins Co., 1946.

[2] AN INTEGRATED PRACTICE OF MEDICINE; A complete General Practice of Medicine from Differential Diagnosis by Presenting Symptoms to Specific Management of the Patient. Vols. 1, 2, 3, 4. By Harold Thomas Hyman, M.D. Philadelphia and London: W. B. Saunders Co., 1946.

[3] FURTHER STUDIES IN ENCEPHALOGRAPHY. By E. Graeme Robertson, M.D., F.R.C.P., F.R.A.C.P. Melbourne: MacMillan & Co., Ltd., 1946.

pages) it is large in scope and contains much practical information supported by experimental evidence on the dynamics of the cerebrospinal fluid in relation to pneumoencephalography. The author presents further observations on the use of his modification of the standard technique of pneumoencephalography as set forth in his previous work on encephalography (MacMillan and Company, Ltd., Melbourne, 1941).

His method is designed to permit the visualization of any selected part of the intracranial cerebrospinal fluid-filled spaces by the use of a minimal quantity of gas. This is achieved by accurate positioning of the patient, especially the head, in relation to the vertical axis. The routes the air takes to enter the ventricles and the various cisternae are detailed by the author from his observations in many cases. The reasons therefore are given in detail both in conclusions drawn from those observations and from most ingenious *in vitro* experiments with analysis of the physical factors involved.

A chapter is devoted to factors which may prevent ventricular filling and one to posture for filling the ventricles. He further believes that his method reduces the risk of encephalography where supratentorial or infratentorial neoplasms cause increased intracranial pressure, while full information is gained.

Special consideration is given to the anatomic arrangement of the subarachnoid cisterns in the posterior fossa from the encephalographic standpoint and to the filling of these with air as well as their demonstration on the x-ray films.

The book is well and profusely illustrated with excellent reproductions of x-rays and tracings of the air shadows noted in them. Many typical histories of special interest problems are given and illustrated with pneumoencephalograms. The differentiations between neoplasm and atrophy, and between neoplasm and degeneration, are discussed, and a chapter is devoted to the developmental abnormalities. This work without doubt, is worthy of the attention of both neurologists and neurosurgeons. HALE HAVEN.

BOOKS RECEIVED

Books received are acknowledged in this department, and such acknowledgment must be regarded as a sufficient return for the courtesy of the sender. Selections will be made for review in the interests of our readers and as space permits.

ELEMENTS OF SURGERY. By Fauset Welsh, B.Sc., M.B., F.R.C.S.(Eng.). London, New York, Toronto: Geoffrey Cumberlege Oxford University Press, 1947.

INFILTRAZIONE NOVOCAINICA DEL PLESSO CELIACO; APPLICAZIONI TERAPEUTICHE. By Renato Rossi. Torino, Italy: Edizioni I.T.E.R., 1945.

MAAGCHIRURGIE. By Dr. A. D. J. M. Verbeek. Amsterdam: L. J. Veen's Uitgeversmaatschappij N.V., 1946.

MEDICAL DISORDERS OF THE LOCOMOTOR SYSTEM INCLUDING THE RHEMATIC DISEASES. By Ernest Fletcher, M.A., M.D. (Cantab.), M.R.C.P. Baltimore: The Williams & Wilkins Co., 1947.

HISTOPATHOLOGY OF THE EAR, NOSE AND THROAT. By Andrew A. Eggston, B.S., M.D. and Dorothy Wolff, A.B., M.A., Ph.D. Baltimore: The Williams & Wilkins Co., 1947.

NEW AND NONOFFICIAL REMEDIES 1947, CONTAINING DESCRIPTIONS OF THE ARTICLES WHICH STAND ACCEPTED BY THE COUNCIL ON PHARMACY AND CHEMISTRY OF THE AMERICAN MEDICAL ASSOCIATION ON JANUARY 1, 1947. Issued Under the Direction and Supervision of the Council on Pharmacy and Chemistry of the American Medical Association. Philadelphia, London, Montreal: J. B. Lippincott Co., 1947.

LES THYROIDECTOMISES. By R. Leibovici and Gilbert-Dreyfus. Paris: G. Doin & Cie, 1946.

1947 RENEWAL PAGES FOR SURGERY OF THE EAR. Samuel J. Kopetzky, M.D., F.A.C.S., Editor. New York: Thomas Nelson & Sons, Publishers, 1947.

EL ENCLAVAMIENTO INTRAMEDULAR DE KÜNTSCHER. By C. Gil Turner. Madrid: Revista Española de Cirugia, 1946.

A MANUAL OF FRACTURES AND DISLOCATIONS. By Barbara Bartlett Stimson, A.B., M.D., Med. Sc.D., F.A.C.S. 2nd ed. Philadelphia: Lea & Febiger, 1947.

OSTEOTOMY OF THE LONG BONES. By Henry Milch, M.D. Springfield, Ill.: Charles C Thomas, 1947.

CONCISE ANATOMY. By Linden F. Edwards, Ph.D. Philadelphia, Toronto: The Blakiston Co., 1947.

CANCER; DIAGNOSIS, TREATMENT, AND PROGNOSIS. By Lauren V. Ackerman, M.D., and Juan A. del Regato, M.D. St. Louis: The C. V. Mosby Co., 1947.

A SHORT HANDBOOK OF PRACTICAL ANAESTHETICS. By Hoel Parry-Price, M.R.C.S., L.R.C.P., D.A. (R.C.S.). Bristol: John Wright & Sons, Ltd.; London: Simpkin Marshall Ltd., 1946.

SIGNS AND SYMPTOMS; THEIR CLINICAL INTERPRETATION. Edited by Cyril Mitchell MacBryde, M.D., F.A.C.P. Philadelphia, London, Montreal: J. B. Lippincott Co., 1947.

CLINICAL CONGRESS OF AMERICAN COLLEGE OF SURGEONS

IRVIN ABELL, Louisville, *President*
ARTHUR W. ALLEN, Boston, *President-Elect*

Committee on Arrangements
HOWARD A. PATTERSON, *Chairman;* FRANK GLENN, *Secretary*

Frank E. Adair
Albert H. Aldridge
William D. Andrus
Thomas M. Brennan
E. Jefferson Browder
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Henry W. Cave
Ralph Colp
Edward J. Donovan
Merrill N. Foote
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Charles A. Gordon
J. William Hinton
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George H. Humphreys
Robert H. Kennedy
William F. MacFee
John H. Mulholland
W. Barclay Parsons
Brittain F. Payne
Otto C. Pickhardt
Raymond P. Sullivan
Howard C. Taylor, Jr.
William Crawford White
Philip D. Wilson
James M. Winfield

PRELIMINARY PROGRAM FOR 1947 CLINICAL CONGRESS, THE WALDORF-ASTORIA, NEW YORK, SEPTEMBER 8 TO 12, 1947

WITH a comprehensive and diversified program of clinics, scientific sessions, hospital conferences, official meetings, surgical films, and educational and technical exhibits, the American College of Surgeons will open its thirty-third annual Clinical Congress in New York on Monday morning, September 8, and continue in session through Friday, September 12. Headquarters will be at The Waldorf- Astoria.

Operative and nonoperative clinics will be held every morning and afternoon in the hospitals of New York and Brooklyn. The preliminary clinical program, and programs for the sessions to be held at The Waldorf-Astoria and the Hotel Commodore, are published in succeeding pages.

The twenty-sixth annual Hospital Standardization Conference will be held as part of the Clinical Congress. Sessions will be held from Monday through Thursday, mornings, afternoons, and evenings, except on Monday evening when the hospital representatives are invited to attend the Presidential Meeting.

The first event on the Clinical Congress program will be the General Assembly for surgeons and hospital representatives which will be held on Monday morning at 10:00 o'clock. The Monday afternoon program will include two panel discussions, one beginning at 1:30, the other at 3:00; clinics in the hospitals; a hospital conference; and a surgical film exhibition. The Presidential Meeting will be held on Monday evening.

The Tuesday morning program will include clinics in the hospitals; the Forum on Fundamental Surgical Problems; a panel discussion on ophthalmology from 9:00 to 10:30; a panel discussion on otorhinolaryngology from 10:45 to 12:15; a hospital conference; and surgical film exhibitions. On Tuesday afternoon there will be clinics in the hospitals; panel discussions from 1:30 to 3:00 and from 3:30 to 5:00; a symposium on fractures and other traumas; a symposium on "Organization for Diagnosis and Treatment of Cancer—Clinics and Detection Centers"; a hospital conference; and surgical film exhibitions. On Tuesday evening there will be three scientific sessions—one on general surgery, one on ophthalmology, and the third on otorhinolaryngology.

The Wednesday and Thursday programs will follow the same pattern except that on Wednesday afternoon a symposium on cancer will replace those on fractures, and on organization of cancer clinics and detection centers, and that on Thursday afternoon these will be replaced by the annual meeting of Fellows at 1:45 and a panel on graduate training in surgery at 3:30 o'clock. On Wednesday evening the ophthalmologists and otorhinolaryngologists will meet in joint session.

The Friday morning program will consist of clinics in the hospitals, the Forum on Fundamen-

tal Surgical Problems, and surgical film exhibitions. The Friday afternoon program will include the Assembly of Initiates; clinics in the hospitals; and six concurrent panel discussions on, respectively, obstetrics, plastic surgery, neurological surgery, thoracic surgery, urology, and orthopedic surgery. The concluding event of the Clinical Congress program will be the Convocation on Friday evening at 8:15 o'clock in the Grand Ballroom.

Detailed programs are listed in succeeding pages.

CLINICAL PROGRAM

The hospitals and medical schools of greater New York are co-operating in scheduling operative and nonoperative clinics, and group clinical conferences. Visiting surgeons will have excellent opportunities to attend well arranged programs of many different kinds in selected hospitals in the area. General and special demonstrations will be held on such subjects as fractures, cancer, maternal morbidity, and end-result studies; clinicopathologic and x-ray conferences; newer diagnostic and therapeutic procedures; preoperative and postoperative supportive treatment; anesthesia; reconditioning; and many other timely subjects. Good opportunities for studying the newer techniques and surgical procedures will be provided.

The program of each general hospital will be arranged to cover subjects in general surgery, obstetrics and gynecology, fractures, orthopedic surgery, thoracic surgery, neurosurgery, genitourinary surgery, ophthalmology, and otolaryngology.

The list of hospitals which are participating in the clinical program, with the names of the hospital representatives, follows:

New York

Beekman-Downtown Hospital—Robert H. Kennedy
Bellevue Hospital
  First Division—Robert Wylie
  Second Division—Ernest Lampe
  Third Division—W. B. Murphy
  Fourth Division—Vansel Johnson
Beth Israel Hospital—Samuel Standard
Flower and Fifth Avenue Hospitals—S. T. Glasser
Goldwater Memorial Hospital—Condict Cutler
Harlem Hospital—David Smith
Harlem Eye and Ear Hospital—C. B. Meding
Hospital for Joint Diseases—A. J. Beller
Hospital for Special Surgery—T. C. Thompson
Lenox Hill Hospital—Otto Pickhardt
Manhattan Eye, Ear and Throat Hospital—Frank Keil and Joseph D. Kelly
Memorial Hospital for the Treatment of Cancer and Allied Diseases—Allen O. Whipple
Metropolitan Hospital—Kenneth C. Peacock
Montefiore Hospital for Chronic Diseases and Country Sanitorium—Arthur H. Aufses
Mt. Sinai Hospital—Ralph Colp
New York Hospital—S. W. Moore
  Lying-in Hospital—Gordon Douglas
New York Eye and Ear Infirmary—Stuart L. Craig
New York Orthopedic Dispensary and Hospital—Halford Hallock
New York Post-Graduate Medical School and Hospital—R. F. Carter
Presbyterian Hospital
  Babies Hospital—Thomas V. Santulli
  Neurological Institute—John Scarff
  Presbyterian Hospital—John Scudder
  Sloane Hospital for Women—Charles Lee Buxton
  Institute of Ophthalmology—Arthur G. DeVoe
  Section on Otolaryngology—James W. Babcock
  Squier Urological Clinic—Charles T. Hazzard
  Presbyterian-New York Orthopedic—Harrison L. McLaughlin
Roosevelt Hospital—James E. Thompson
St. Luke's Hospital—Paul C. Morton
St. Vincent's Hospital—Raymond P. Sullivan
U. S. Marine Hospital—Homer L. Skinner
Veterans Administration Hospital—Paul K. Sauer
Woman's Hospital—Ralph L. Barrett

Brooklyn

Brooklyn Eye and Ear Hospital—M. A. Lasky
Cumberland Hospital—Herbert Wikle
House of St. Giles, The Cripple—C. C. Vitale
Jewish Hospital—Louis Berger
Kings County Hospital—Joseph Tenopyr
  Long Island College Division—Philip Lear
Long Island College Hospital—Albert Ritzmann
Methodist Hospital—P. A. Renaud
St. John's Hospital—Merrill N. Foote

HOSPITAL STANDARDIZATION CONFERENCE

The first formal session of the Clinical Congress will be the opening meeting of the twenty-sixth Hospital Standardization Conference. Dr. Irvin Abell of Louisville, President of the American College of Surgeons, will preside. The hospital conferences will continue on Monday afternoon, with sessions following on Tuesday, Wednesday and Thursday mornings, afternoons and evenings.

Hospital administrators, members of governing boards, medical staff members, heads of the various hospital departments and their personnel, nurses, dietitians, medical records librarians, and many other persons directly concerned about hospital progress, will be interested in the discussions of current hospital problems.

The Monday afternoon session will be devoted to a panel discussion on current problems in medical service in hospitals; Tuesday morning to improvement in food service in hospitals; Tuesday afternoon to improvement in nursing service in hospitals; Tuesday evening to a joint session for hospital trustees, medical staff officers, and administrators; Wednesday morning to discussion of personnel and public relations; Wednesday afternoon to a joint session with the American Association of Medical Record Librarians on improving medical records; Wednesday evening to a

forum on trends in hospital administration and special hospital problems; Thursday morning to a forum on special problems; Thursday afternoon to a round table conference on problems of the small hospital in meeting the standards of the American College of Surgeons and to a symposium on Graduate Training in Surgery; and Thursday evening to a special conference on the point rating system.

Detailed programs appear on succeeding pages.

ANNUAL MEETING

The annual meeting of the Governors and Fellows will be held on Thursday afternoon at 1:45. There will be election of officers and governors. The annual meeting affords the Fellows of the College an opportunity to hear reports of officials on the work of the organization, and to broaden their knowledge of the methods, and their effectiveness, which are used to raise the professional and ethical standards of surgery, and to promote good hospital care and general improvement in the practice of medicine. Each Fellow has a personal part in this work and may extend the influence of the College materially in his local community and wherever his contacts extend. Hospital Standardization particularly offers him wide opportunity to provide better medical care for his patients in the hospital in which he works, through continuous progress in applying the principles of the Minimum Standard which insure the best care of the patient.

Every Fellow will be interested in the reports which will be presented on the following subjects: finances; Hospital Standardization; Graduate Training in Surgery; Medical Motion Pictures; Publications; Public Relations; Library and Literary Research; the work of the state and provincial credentials committees, committees on applicants, and the Committee on History Reviews; Sectional Meetings, 1947; and the Department of Clinical Research, including cancer clinics and cancer detection centers, the Committee on Cancer, Medical Service in Industry, and the Committee on Fractures and other Traumas. Dr. Abell, Chairman of the Board of Regents, will report on administration of the College and Dr. Arthur W. Allen will discuss the obligations and opportunities of the Fellows.

MEDICAL MOTION PICTURES

The latest available films demonstrating surgical procedures and related subjects will be shown in the medical motion picture exhibits which will be held daily in The Waldorf-Astoria. Motion pictures are a much appreciated feature of the Clinical Congress. Many new pictures on varied subjects are being received by the American College of Surgeons for review. Both sound and silent colored films will be shown during the Congress, all of which will have been approved by the Committee on Medical Motion Pictures.

There will also be shown the first of a series of films which are being produced by the College under a grant from the Johnson & Johnson Research Foundation. The film is entitled, "Anomalies of the Bile Ducts and Blood Vessels; Strictures of the Common Duct," a teaching film of which Dr. Warren H. Cole of Chicago is the author. It is contemplated that there will also be completed in advance of the Clinical Congress at least one additional film which is being produced by the College under the expanded program.

ADVANCE REGISTRATION

The hospitals and medical schools of New York afford accommodations for a large number of visiting surgeons. However, in order to insure against overcrowding, attendance at the Congress will be limited to the number that can be accommodated at the clinics and meetings. It is therefore expected that surgeons who wish to attend the Congress will register in advance.

Because of greatly increased costs and expenses to which the College will be subjected this year in connection with its New York Clinical Congress, the Board of Regents has found it necessary to require a registration fee of $5.00 for Fellows and for endorsed Junior Candidates. Non-Fellows attending as invited guests of the College will pay a fee of $10.00. No fee will be required of initiates of the class of 1947.

The registration desk will be open from 3:00 to 9:00 p.m. on Sunday, September 7, in order to permit surgeons to obtain tickets for the Monday clinics. A new plan is to be adopted in New York of distributing clinic tickets on the day prior to the clinics, from 9:00 a.m. to 5 p.m. Any remaining tickets will be available at 7:00 a.m. the same day on which the clinics are held.

TECHNICAL AND SCIENTIFIC EXHIBITION

The technical exhibit, together with the registration and clinic ticket bureaus, will be located in the Basildon Room, Jade Room, and Astor Gallery, all on the third floor of the hotel. Leading manufacturers of surgical instruments, x-ray apparatus, sterilizers, operating room lights, ligatures, dressings, hospital apparatus and supplies of all kinds, and pharmaceuticals, and publishers of medical books will be represented. The technical exhibits will demonstrate many of the new features learned from our experience in the war.

GENERAL ASSEMBLY

JOINT SESSION—SURGEONS AND HOSPITAL REPRESENTATIVES

Monday, 10:00 a.m.–12:30 p.m.—Grand Ballroom—The Waldorf-Astoria

IRVIN ABELL, M.D., Louisville; President, American College of Surgeons, Presiding.
Welcome from the Mayor and the Hospitals of New York. EDWARD M. BERNECKER, M.D., New York; Commissioner, Department of Hospitals, City of New York.
Greetings from the American Hospital Association. JOHN H. HAYES, New York; President.
Greetings from the Canadian Hospital Council. HARVEY AGNEW, M.D., Toronto; Secretary.
Summary of College Activities as Related to Hospitals for Past Year. IRVIN ABELL, M.D., Chairman, Board of Regents.
Need for Efficient Medical Staff Organization and Control of the Professional Work of the Hospital. HAROLD L. FOSS, Danville, Pennsylvania; Surgeon-in-Chief, Geisinger Memorial Hospital.
Better Hospital Facilities for Rural Patients—Plans to Integrate Small General Hospitals with Nearest Institutions Incorporating Specialized Services. GRAHAM L. DAVIS, Battle Creek; Hospital Director, W. K. Kellogg Foundation; President-Elect, American Hospital Association.
The New Era in Personnel Relations and its Meaning for Hospitals. BRIGADIER GENERAL ROBERT W. JOHNSON, New Brunswick; Chairman, Board of Trustees, Johnson & Johnson.
The Potentialities of the Medical Motion Picture for Teaching Purposes. WARREN H. COLE, M.D., Chicago; Professor of Surgery and Head of the Department, University of Illinois College of Medicine in collaboration with TOM JONES, Chicago; Professor Medical Illustration, University of Illinois College of Medicine.
Showing: "Anomalies of the Bile Ducts and Blood Vessels; Strictures of the Common Duct."

PRESIDENTIAL MEETING

Monday, 8:15–10:30 p.m.—Grand Ballroom, The Waldorf-Astoria

IRVIN ABELL, M.D., Louisville; President, American College of Surgeons, Presiding.
Processional—Officers, Regents, and Distinguished Guests.
Address of Welcome. HOWARD A. PATTERSON, M.D., New York; Chairman, Committee on Arrangements.
Introduction of Distinguished Guests. ARTHUR W. ALLEN, M.D., Boston; Vice Chairman, Board of Regents.
Address of the Retiring President: The Spirit of Surgery. IRVIN ABELL, M.D., Louisville.
Inauguration of Officers:
Presented by: LELAND S. MCKITTRICK, M.D., Boston; Retiring First Vice President.
President: ARTHUR W. ALLEN, M.D., Boston.
First Vice President: THOMAS E. JONES, M.D., Cleveland.
Second Vice President: GORDON B. NEW, M.D., Rochester, Minnesota.
The Second Martin Memorial Lecture: The Qualifications of the Surgeon and the Cancer Problem. ALLEN O. WHIPPLE, M.D., D.Sc., New York; Clinical Director, Memorial Hospital; Emeritus Valentine Mott Professor of Surgery, Columbia University College of Physicians and Surgeons.
Recessional.

CONVOCATION

Friday, 8:15–10:30 p.m.—Grand Ballroom, The Waldorf-Astoria

ARTHUR W. ALLEN, M.D., Boston; President, American College of Surgeons, Presiding.
Processional: Initiates, Officers, Regents, and Distinguished Guests.
Presentation of Initiates for Fellowship. IRVIN ABELL, M.D., Louisville; Chairman, Board of Regents.
Fellowship Pledge. Recital by Initiates.
Conferring of Fellowships by the President. ARTHUR W. ALLEN, M.D., Boston.
Conferring of Honorary Fellowships. The President.
Fellowship Address: Liberal Medical Education. ANDREW C. IVY, M.D., Chicago; Vice President, University of Illinois, in charge of Chicago Professional Colleges, and Distinguished Professor of Physiology.
Recessional.
Reception by the Officers and Regents for the Initiates and Fellows.

ASSEMBLY OF INITIATES

Friday, 1:45–3:00 p.m.

IRVIN ABELL, M.D., Louisville; Chairman of the Board of Regents, Presiding.
The Program of the American College of Surgeons: Hospital Standardization, Sectional Meetings, Medical Motion Pictures, Library and Literary Research, Graduate Training in Surgery. MALCOLM T. MACEACHERN, M.D., Chicago; Associate Director and Chairman of the Administrative Board.
Fellowship in the American College of Surgeons. IRVIN ABELL, M.D., Louisville.
The Department of Clinical Research of the American College of Surgeons. CHARLES R. BRANCH, M.D., Chicago, Assistant Director.
Qualifications for Fellowship. H. P. SAUNDERS, M.D., Chicago, Assistant Director.

EVENING SCIENTIFIC SESSIONS

GENERAL SURGERY

Tuesday, 8:00–10:30 p.m.

Symposium on The Proper Use of Blood and Blood Plasma

A Permanent Red Cross Transfusion Service. G. FOARD MCGINNES, M.D., Washington.
Blood Bank Organization. PAUL I. HOXWORTH, M.D., Cincinnati.
Reactions to Blood Transfusion and the Rh Factor. LOUIS K. DIAMOND, M.D., Boston.
The Use of Blood and Plasma in Surgery. DALLAS B. PHEMISTER, M.D., Chicago.

Wednesday, 8:00–10:30 p.m.

Fracture Oration. FRANK D. DICKSON, M.D., Kansas City.

Symposium on Nutritional Requirements in Surgery

Proper Employment of Oral Feeding. EVERETT I. EVANS, M.D., Richmond.
Parenteral Feeding. ROBERT ELMAN, M.D., St. Louis.
The Importance of Adequate Supply of Blood and Nourishment for Wound Healing. S. CHAMP LYONS, M.D., New Orleans.

Thursday, 8:00–10:30 p.m.

Symposium on Hypertension

Fundamental Physiological Considerations. PETER HEINBECKER, M.D., St. Louis.
Indications for Surgery in the Treatment of Hypertension. THOMAS FINDLEY, M.D., New Orleans.
Surgical Procedures in the Treatment of Hypertension. REGINALD H. SMITHWICK, M.D., Boston.
End-Results in the Treatment of Hypertension. CYRUS C. STURGIS, M.D., Ann Arbor.

OPHTHALMOLOGY

Tuesday, 8:00–10:30 p.m.

Epithelial Downgrowth Following Cataract Operation. MILTON L. BERLINER, M.D., New York.
Local Anesthesia in Ophthalmology. WALTER S. ATKINSON, M.D., Watertown, New York.
Operative Treatment of Vertical Tropias. BRITTAIN F. PAYNE, M.D., New York.
Prevention and Management of Operative Complications During Cataract Extraction. JACK S. GUYTON, M.D., Baltimore.

Thursday, 8:00–10:30 p.m.

Prevention of Infection in Eye Surgery. M. HAYWARD POST, M.D., St. Louis.
Glaucoma Following Cataract Extraction. WILLIAM C. OWENS, M.D., Baltimore.
Retinal Detachment. HARVEY E. THORPE, M.D., Pittsburgh.
The Difficult Cataract Extraction. R. TOWNLEY PATON, M.D., New York.

OTORHINOLARYNGOLOGY

Tuesday, 8:00–10:00 p.m.

Symposium on Bronchiectasis

Relationship of Bronchiectasis to Sinusitis. ROBERT L. GOODALE, M.D., Boston.
The Surgical Aspects of Bronchiectasis. ALTON OCHSNER, M.D., New Orleans.
The Use of Bronchoscopy in Bronchiectasis. FLETCHER D. WOODWARD, M.D., Charlottesville.

Thursday, 8:00–10:30 p.m.

Symposium on Management of Carcinoma of the Larynx

Treatment by Laryngofissure. CHEVALIER L. JACKSON, M.D., Philadelphia.
Laryngectomy. LOUIS H. CLERF, M.D., Philadelphia.
Management of Carcinoma of the Larynx with Metastatic Involvement of the Neck. HENRY B. ORTON, M.D., Newark.

COMBINED SESSION ON OPHTHALMOLOGY-OTORHINOLARYNGOLOGY

Wednesday, 8:00–10:30 p.m.

Blood Supply of Eye and Sinuses. OSCAR V. BATSON, M.D., Philadelphia.
Dacryocystorhinostomy. HAROLD GIFFORD, Jr., M.D., Omaha.
Relation of the Nasal Accessory Sinuses to Eye Diseases. EDWARD A. LOOPER, M.D., Baltimore.
Ophthalmoneurologic Symptoms of Nasopharyngeal Tumors. FRANK B. WALSH, M.D., Baltimore.

FORUM ON FUNDAMENTAL SURGICAL PROBLEMS

Tuesday through Friday, 8:30 a.m. to 12:30 p.m.—The Waldorf-Astoria

Presentation of the results of clinical and experimental research on problems related to general surgery and the surgical specialties which are being currently conducted in many medical schools, clinics, and hospitals.

Tuesday, September 9, 8:30 a.m.–12:30 p.m.

Gastric Surgery, *Grand Ballroom.*
Wound Healing, Plastic Surgery, and Infections, *Sert Room.*

Wednesday, September 10, 8:30 a.m.–12:30 p.m.

Surgery of the Small and Large Bowel, *Grand Ballroom.*
Protein, Fluid, and Electrolyte Balance, *Sert Room.*

Thursday, September 11, 8:30 a.m.–12:30 p.m.

Gall-bladder, Pancreas, and Peritoneum, *Grand Ballroom.*
Surgery of the Heart and Blood Vessels, *Wedgewood Room.*

Friday, September 12, 8:30 a.m.–12:30 p.m.

Thoracic Surgery, Anesthesia, and Thyroid, *Grand Ballroom.*
Neurosurgery, Tumors, and Urology, *Sert Room.*

SYMPOSIUM ON CANCER CLINICS AND CANCER DETECTION CENTERS

Tuesday 2:00–5:00 p.m.

GRANTLEY W. TAYLOR, M.D., Boston, Chairman, Cancer Committee, American College of Surgeons, presiding.
Early Diagnosis of Carcinoma of Stomach. FORDYCE B. ST. JOHN, M.D., PAUL C. SWENSON, M.D., HAROLD D. HARVEY, M.D., New York.
Experience with the Chicago Cancer Detection Center. AUGUSTA WEBSTER, M.D., Chicago.
The U.S.P.H.S. Cancer Control Program. AUSTIN V. DIEBERT, M.D., Bethesda.
Utilization of Cancer Clinics for Teaching Purposes:
For Undergraduates and Interns in a University Clinic. MAX TAFFEL, M.D., New Haven.
For Members of the Hospital Staff in a Clinic Not Affiliated with a University. WILLIAM R. LAIRD, M.D., Montgomery, West Virginia.
Utilization of Cancer Records from Clinics and Hospitals. FRANK E. ADAIR, M.D., New York

SYMPOSIUM ON CANCER

Wednesday, 2:00–5:00 p.m.

GRANTLEY W. TAYLOR, M.D., Boston, Chairman, Cancer Committee, American College of Surgeons, Presiding.
Surgical Treatment of Carcinoma of the Lung. MICHAEL E. DEBAKEY, M.D., New Orleans.
Tumors of the Testis, ROBERT A. MOORE, M.D., St. Louis
Biological Differentiation of Benign and Malignant Tumors. HARRY S. N. GREENE, M.D., New Haven.
Isotopes in Surgical Research. FRANCIS D. MOORE, M.D., Boston.
Prognosis in Carcinoma of the Bowel. R. KENNEDY GILCHRIST, M.D., Chicago.
Anterior Resection of Carcinoma of the Colon and Rectum. OWEN H. WANGENSTEEN, M.D., Minneapolis.

PANEL DISCUSSIONS

GENERAL SURGERY

Monday, 1:30–3:00 p.m.

Surgery of Pulmonary Infections

Moderator: EVARTS A. GRAHAM, M.D., St. Louis.

Collaborators: BRIAN B. BLADES, M.D., Washington; RICHARD H. OVERHOLT, M.D., Brookline, Massachusetts; HAROLD NEUHOF, M.D., New York.

Monday, 3:30–5:00 p.m.

Pancreatitis

Moderator: MIMS GAGE, M.D., New Orleans.

Collaborators: JOHN M. WAUGH, M.D., Rochester, Minnesota; HORACE J. MCCORKLE, M.D., San Francisco.

Tuesday, 1:30–3:00 p.m.

Surgery of the Spleen

Moderator: ALLEN O. WHIPPLE, M.D., New York.

Collaborators: GEORGE M. CURTIS, M.D., Columbus; ARTHUR H. BLAKEMORE, M.D., New York; ROBERT H. E. ELLIOTT, M.D., New York; LOUIS M. ROUSSELOT, M.D., New York.

Tuesday, 3:30–5:00 p.m.

Jaundice

Moderator: CHARLES B. PUESTOW, M.D., Chicago.

Collaborators: I. S. RAVDIN, M.D., Philadelphia; JAMES T. PRIESTLY, M.D., Rochester, Minnesota; ROSCOE R. GRAHAM, M.D., Toronto.

Wednesday, 1:30–3:00 p.m.

Gastric Surgery

Moderator: HOWARD K. GRAY, M.D., Rochester, Minnesota.

Collaborators: GEORGE T. PACK, M.D., New York; WARREN H. COLE, M.D., Chicago; LELAND S. MCKITTRICK, M.D., Boston.

Wednesday, 3:30–5:00 p.m.

Use of Antibiotic Agents and Chemotherapy in Surgery

Moderator: JOHN S. LOCKWOOD, M.D., New York.

Collaborators: FRANK L. MELENEY, M.D., New York; C. PHILLIP MILLER, M.D., Chicago; HAROLD A. ZINTEL, M.D., Philadelphia; MAJOR EDWIN J. PULASKI, M.C., Fort Sam Houston, Texas.

Thursday, 3:30–5:00 p.m.

Surgery of the Hand

Moderator: HENRY C. MARBLE, M.D., Boston.

Collaborators: HARVEY S. ALLEN, M.D., Chicago; LAURIE H. MCKIM, M.D., Montreal; J. HAROLD COUCH, M.D., Toronto.

UROLOGY

Friday, 1:30–4:30 p.m.

Moderator: CHARLES C. HIGGINS, M.D., Cleveland.

Treatment of Carcinoma of the Bladder.

Collaborators: HUGH J. JEWETT, M.D., Baltimore; ARCHIE L. DEAN, M.D., New York; EDWARD N. COOK, M.D., Rochester, Minnesota.

Management of Renal Lithiasis

Collaborators: GEORGE F. CAHILL, M.D., New York; LINWOOD D. KEYSER, M.D., Roanoke; RICHARD W. SATTERTHWAITE, M.D., Battle Creek.

ORTHOPEDIC SURGERY

Moderator: PAUL B. MAGNUSON, M.D., Washington.

Problem of Fractures of the Neck of the Femur

Collaborators: KELLOGG SPEED, M.D., Chicago; RUDOLPH S. REICH, M.D., Cleveland; PAUL C. COLONNA, M.D., Philadelphia.

Indications and Operative Technics for Lumbosacral Fusion of the Spine

Collaborators: GUY A. CALDWELL, M.D., New Orleans; DAVID M. BOSWORTH, M.D., New York; MARK B. COVENTRY, M.D., Rochester, Minnesota.

GYNECOLOGY AND OBSTETRICS

Moderator: J. Mason Hundley, Jr., M.D., Baltimore.

Changes in the Urinary System Engendered by Gynecological and Obstetrical Conditions

Collaborators: Houston Spencer Everett, M.D., Baltimore; William E. Studdiford, Jr., M.D., New York; R. Gordon Douglas, M.D., New York.

Modern Trends in the Treatment of Malignancies of the Female Genitalia

Collaborators: Nelson B. Sackett, M.D., New York; Robert A. Ross, M.D., Durham; William P. Healy, M.D., New York.

NEUROLOGICAL SURGERY

Late Results of Compound Craniocerebral Injuries

Moderator: A. Earl Walker, M.D., Chicago.

Collaborators: James C. White, M.D., Boston; Cobb Pilcher, M.D., Nashville; Michael Scott M.D., Philadelphia.

Surgical Aspects of Vascular Disorders of the Brain

Moderator: James L. Poppen, M.D., Boston.

Collaborators: Wallace B. Hamby, M.D., Buffalo; Winchell Mck. Craig, M.D., Rochester; W. James Gardner, M.D., Cleveland.

THORACIC SURGERY

Moderator: Stuart W. Harrington, M.D., Rochester, Minnesota.

Surgery of the Esophagus

Collaborators: Herman J. Moersch, M.D., Rochester, Minnesota; Richard H. Sweet, M.D., Boston, Carl Eggers, M.D., New York; Orvar Swenson, M.D., Boston.

Tumors of the Lung, Benign and Malignant

Collaborators: (Same as above)

PLASTIC SURGERY

Moderator: William G. Hamm, M.D., Atlanta.

Repair of Surface Defects of the Extremities

Collaborators: Truman G. Blocker, Jr., M.D., Galveston; Frank McDowell, M.D., St. Louis; W. Brandon Macomber, M.D., Albany.

Major Facial Defects

Collaborators: Bradford Cannon, M.D., Boston; W. Bowdoin Davis, M.D., Baltimore; Carl E. Lischer, M.D., St. Louis; Major Andrew M. Moore, M.C., Phoenixville.

OPHTHALMOLOGY

Tuesday, 9:00–10:30 a.m.

Surgery of Extraocular Muscles

Moderator: Harold W. Brown, M.D., New York.

Collaborators: Rudolf Aebli, M.D., New York; Walter H. Fink, M.D., Minneapolis; Richard G. Scobee, St. Louis.

Wednesday, 9:00–10:30 a.m.

Surgery of Glaucoma

Moderator: C. S. O'Brien, M.D., Iowa City.

Collaborators: P. Robb McDonald, M.D., Philadelphia; John S. McGavic, M.D., Bryn Mawr; Willis S. Knighton, M.D., New York.

Thursday, 9:00–10:30 a.m.

Angiospastic Ocular Lesions

Moderator: Frank D. Carroll, M.D., New York.

Collaborators: Walter F. Duggan, M.D., Utica; Herman Elwyn, M.D., New York; Henry P. Wagener, M.D., Rochester, Minnesota.

OTORHINOLARYNGOLOGY

Tuesday, 10:45 a.m.–12:15 p.m.

The Use of Antibiotics in Ear, Nose and Throat Surgery

Moderator: Chester S. Keefer, M.D., Boston.

Collaborators: Westley M. Hunt, M.D., New York; Frederick T. Hill, M.D., Waterville, Maine; Leighton F. Johnson, M.D., Boston.

Wednesday, 10:45 a.m.–12:15 p.m.

Rehabilitation of the Larynx in Bilateral Adductor Paralysis of the Vocal Cords

Moderator: GABRIEL TUCKER, M.D., Philadelphia.

Collaborators: DEGRAAF WOODMAN, M.D., New York; BRIEN T. KING, M.D., Seattle; ALBERT C. FURSTENBERG, M.D., Ann Arbor.

Thursday, 10:45 a.m.–12:15 p.m.

Anesthesia for Ear, Nose and Throat Surgery

Moderator: LLOYD H. MOUSEL, M.D., Washington.

Collaborators: CARL H. MCCASKEY, M.D., Indianapolis; PETER N. PASTORE, M.D., Richmond; CURTISS B. HICKCOX, M.D., Philadelphia; PERRY P. VOLPITTO, M.D., Augusta.

SYMPOSIUM ON FRACTURES AND OTHER TRAUMAS

Tuesday, 2:00–5:00 p.m.

ROBERT H. KENNEDY, M.D., New York; Chairman, Committee on Fractures and Other Traumas, Presiding.

Appraisal of Present Methods of Treatment

Intracapsular Fractures of the Neck of the Femur. PAUL C. COLONNA, M.D., Philadelphia; Professor of Orthopedic Surgery, University of Pennsylvania School of Medicine.

Fractures of the Upper Extremity of the Tibia Involving the Knee Joint. EDWIN F. CAVE, M.D., Boston; Instructor in Orthopedic Surgery, Harvard Medical School.

Heavy Resistance Exercise in Orthopaedics. THOMAS L. DELORME, Jr., M.D., Boston.

Injuries of the Intervertebral Disc. FRANK H. MAYFIELD, M.D., Cincinnati; Assistant in Clinical Surgery, University of Cincinnati College of Medicine.

Fractures of the Os Calcis. JAMES J. CALLAHAN, M.D., Chicago; Professor, Bone and Joint Surgery, Loyola University School of Medicine.

Bone Plates and Screws. ROBERT H. KENNEDY, M.D., New York; Clinical Professor of Surgery, New York University College of Medicine (Postgraduate Division).

SYMPOSIUM ON GRADUATE TRAINING IN SURGERY AND THE SURGICAL SPECIALTIES

Thursday, 3:30–5:30 p.m.

DALLAS B. PHEMISTER, M.D., Chicago; Chairman, Committee on Graduate Training in Surgery, American College of Surgeons, Presiding.

Centralized Plan of Graduate Training in Surgery. MAX M. ZINNINGER, M.D., Cincinnati; Associate Professor of Surgery, University of Cincinnati College of Medicine.

Graduate Training from the Standpoint of Decentralized Residency Training in Hospitals Affiliated with a Medical School. ALBERT C. FURSTENBERG, M.D., Ann Arbor; Professor of Otolaryngology, Chairman of the Department of Otolaryngology and Dean of the University of Michigan Medical School.

The Place of the Organized Clinic in Graduate Training in Surgery. RICHARD B. CATTELL, M.D., Boston; Surgeon, Lahey Clinic.

Graduate Training in General Surgery and the Surgical Specialties in a Veterans Hospital. CHARLES B. PUESTOW, M.D., Chicago; Clinical Professor of Surgery, University of Illinois; Deputy Chiefof Surgery, Veterans Administration Hospital, Hines.

Discussion: Opened by FREDERICK A. COLLER, M.D., Ann Arbor; Professor of Surgery, Chairman of the Department of Surgery, University of Michigan Medical School; Chairman, Committee on Relations, American College of Surgeons with the American Boards, and FRASER B. GURD, M.D., Montreal; Professor of Surgery, McGill University Faculty of Medicine.

HOSPITAL STANDARDIZATION CONFERENCE

GENERAL ASSEMBLY: JOINT SESSION—SURGEONS AND HOSPITAL REPRESENTATIVES

Monday, 10:00 a.m.–12:30 p.m. (See program on preceding pages.)

CURRENT PROBLEMS IN MEDICAL SERVICE IN HOSPITALS

Monday, 2:00–5:00 p.m.—Sert Room—First Floor—The Waldorf-Astoria

Conducted by JOSEPH TURNER, M.D., New York, Director, Mount Sinai Hospital.

Integration of the General Practitioner in the Hospital Medical Staff Organization. HARRY R. MENDELSOHN, M.D., Cincinnati; President, The Society of General Physicians.

The Professional Audit to Control Efficiency. FREDERICK T. HILL, M.D. Waterville, Maine; Medical Director, Thayer Hospital.

What is Major and Minor Surgery? Who Should be Permitted to do Major Surgery in an Approved Hospital? J. J. GOLUB, M.D., New York; Director, Hospital for Joint Diseases.

Medical Staff Conferences Essential to Medical Progress and Good Care of the Patient. JOSEPH C. DOANE, M.D., Philadelphia; Medical Director, Jewish Hospital and Professor of Clinical Medicine, Temple University Medical School; Professor of Medicine, Graduate School of Medicine, University of Pennsylvania.

Round Table Conference on Medical Staff Problems. Moderator: EVERETT W. JONES, Chicago; Vice-President, Modern Hospital Publishing Company.

8:00–10:30 p.m.—Ballroom—Fourth Floor—The Waldorf Astoria
Presidential Meeting

Hospital representatives attending the Twenty-Sixth Annual Hospital Standardization Conference are most cordially invited to attend the Presidential Meeting.

IMPROVING FOOD SERVICE IN HOSPITALS

Tuesday, 9:30 a.m.–12:30 p.m.—Grand Ballroom—Hotel Commodore

Conducted by MARGARET GILLAM, Chicago; Dietary Consultant, American Hospital Association.

New Developments in the Field of Nutrition and How They Affect Food Service and the Care of the Surgical Patient. ROBERT ELMAN, M.D., St. Louis; Professor of Clinical Surgery, Washington University School of Medicine.

New Equipment and Methods to Relieve Problems of Food Service Conducive to Complaints and Dissatisfaction on the Part of the Patient. EVERETT W. JONES, Chicago; Vice-President, The Modern Hospital Publishing Company.

The Importance of Good Interdepartmental Relations of the Dietary Department and How They can be Improved. ANTHONY W. ECKERT, Neptune, New Jersey; Administrator, Fitkin Memorial Hospital.

Personnel Problems in the Dietary Department. E. ALLIENE MOSSO, New York; Chief Dietitian, St. Luke's Hospital.

Food Service an Important Factor in Personnel and Public Relations. PAUL H. FESLER, Oklahoma City; Administrator, University of Oklahoma Hospitals.

Question and Answer Period.

Problems of Food Service in Hospitals.

IMPROVING NURSING SERVICE IN HOSPITALS

Tuesday, 2:00–5:00 p.m.—Sert Room—First Floor—The Waldorf-Astoria

Conducted by CLAUDE W. MUNGER, M.D., New York; Director, St. Luke's Hospital and Professor of Hospital Administration, Columbia University.

Report of Survey Made by American College of Surgeons on use of Auxiliary Workers in Nursing Service. HOWARD C. NAFFZIGER, M.D., San Francisco; Professor of Surgery, University of California Medical School; Surgeon-in-Chief, University Hospital.

Utilization and Training of Practical Nurses and Auxiliary Workers. ELISABETH C. PHILLIPS, R.N., Rochester, New York; Executive Director, Visiting Nurse Association; Chairman, Joint Committee on Auxiliary Nursing Service.

How Hospitals Can Help in Student and Practical Nurse Recruitment. EMILY K. JOHNSON, New York; Public Relations Associate, Nursing Information Bureau, American Nurses' Association.

Correlation of the Services of the Graduate Nurse, the Practical Nurse, and the Nurses' Aide. EDWARD M. BERNECKER, M.D., New York; Commissioner, Department of Hospitals, New York.

Discussion—Questions and Answers *conducted by* CLAUDE W. MUNGER, M.D., Chairman.

JOINT SESSION FOR HOSPITAL TRUSTEES, MEDICAL STAFF OFFICERS, AND ADMINISTRATORS

Tuesday, 8:00–10:30 p.m.—Sert Room—First Floor—The Waldorf-Astoria

RAYMOND P. SLOAN, New York; Editor, The Modern Hospital, Presiding.

Hospitals Need Leadership. RAYMOND P. SLOAN, New York.

Working Together—Hospital Trustees, Members of the Medical Staff and the Administrator, Must Co-operate in Assuring Good Care of the Patient in the Hospital. JOHN H. HAYES, New York; Superintendent, Lenox Hill Hospital; President, American Hospital Association.

Regionalization of Hospitals. EDWARD J. NOBLE, New York; Chairman of the Board, American Broadcasting Company, and Chairman, Life Savers Corporation.

A New Era in the Education and Training of Hospital Administrators. CLAUDE W. MUNGER, M.D., New York; Director, St. Luke's Hospital; Professor of Hospital Administration, Columbia University.

Question and Answer period.

IMPROVING PERSONNEL RELATIONS AND PUBLIC RELATIONS

Wednesday, 9:30 a.m.–12:30 p.m.—Grand Ballroom Hotel Commodore

Conducted by LEO M. LYONS, Chicago; Executive Director, St. Luke's Hospital.

Better People Make Better Hospitals—Good Personnel Relations Can Improve Quality of Personnel. EDGAR C. HAYHOW, East Orange, New Jersey; Director, East Orange General Hospital; President-Elect, American College of Hospital Administrators.

Personnel Relations Affect Public Relations. OLIVER G. PRATT, Providence; Executive Director, Rhode Island Hospital.

Why Every Hospital Should Have—(a) A Personnel Director and Definite Personnel Policies, (b) Some Person Definitely Assigned Over-all Responsibility for Public Relations. FRASER D. MOONEY, M.D., Buffalo; Superintendent, Buffalo General Hospital; Associate in Medicine, University of Buffalo School of Medicine.

Orientation and Training of Hospital Personnel. MARY A. JOHNSON, New York. Research Associate, Hospital Administration, School of Public Health, Columbia University.

Why Hospitals Must Consciously Enter Competition for the Public's Attention and What Means They Can Ethically Employ. CORNELIUS M. SMITH, New York; President, Will, Folsom and Smith.

Round Table Conference on Media for Informing the Public Regarding Hospitals.

Newspapers	Films and Slides
Radio	Forums and Talks
Hospital Publications	Tours and Demonstrations

Conducted by JOHN GORRELL, M.D., New York; Associate Professor of Hospital Administration, Columbia University. School of Medicine.

IMPROVING MEDICAL RECORDS IN HOSPITALS—JOINT SESSION WITH THE AMERICAN ASSOCIATION OF MEDICAL RECORD LIBRARIANS

Wednesday, 2:00–5:00 p.m.—Sert Room First Floor—The Waldorf-Astoria

Conducted by ROBIN C. BUERKI, M.D., Philadelphia; Director of Hospitals, University of Pennsylvania; Dean, Graduate School of Medicine.

A Message from the President of the American Association of Medical Record Librarians. ENNA C. BLACK, R.R.L., New Haven; Medical Record Librarian, Grace Unit of the Grace-New Haven Community Hospital; President of the American Association of Medical Record Librarians.

The Present Status of Training Medical Record Librarians Through—

(a) Approved Schools. EDNA K. HUFFMAN, R.R.L., Chicago; Director, School for Medical Record Librarians, Wesley Memorial Hospital; Director, Program in Medical Record Library Science, Northwestern University.

(b) In-Service Training Institute. MARGARET C. TAYLOR, R.R.L. Chicago; Field Instructor, Extension Course, American Association of Medical Librarians.

The Proper Approach to the Maintaining of Adequate Medical Records. MARGARET DUBOIS, M.D., Richmond; Assistant Director, Hospital Division, and Associate Professor of Hospital Administration, Medical College of Virginia School of Medicine.

The Responsibility of the Medical Staff of the Hospital in Maintaining Good Medical Records. MALCOLM T. MACEACHERN, M.D., Chicago; Associate Director, American College of Surgeons.

Impressions of the Field Representative of the American College of Surgeons in Regard to Maintaining Adequate Medical Records. REAR ADMIRAL LUCIUS W. JOHNSON, San Diego; Assistant, Hospital Department, Assigned to Pacific Region.

Question and Answer Period. Conducted by ROBIN, C. BUERKI, M.D., Chairman.

A FORUM ON TRENDS IN HOSPITAL ADMINISTRATION, NEW IDEAS AND PROCEDURES, AND SPECIAL HOSPITAL PROBLEMS

Wednesday, 8:00–10:30 p.m.—Sert Room—First Floor—The Waldorf-Astoria

Conducted by FRANK R. BRADLEY, M.D., St. Louis; Director, Barnes Hospital; President, American College of Hospital Administrators.

Opportunity has been given to the graduates and administrative interns of the various Universities now offering courses in hospital administration to present summary transcripts of new ideas and trends in hospital work, including planning and construction, improved equipment, new procedures and technics, administrative problems, personnel management, public relations and other phases of hospital administration. From the briefs or transcripts presented ten ten-minute presentations will be selected for discussion at this session.

FORUM ON SPECIAL PROBLEMS

Thursday, 9:30 a.m —12:30 p.m.—Sert Room—First Floor—The Waldorf-Astoria

Coordinator: ROBIN C. BUERKI, M.D., Philadelphia; Director of Hospitals, University of Pennsylvania; Dean, Graduate School of Medicine.

Topics for Discussion:

The Modern Hospital—a Complete Synchronized Entity. New Developments in Each Group of Departments that Provide Unified Service to the Patient.

What Has Been Done to Meet the Present Acute Bed Shortage?

Present Status of Volunteer Workers in Hospitals.

Hospital Trustees, Members of the Medical Staff, Nursing Executives and Administrators Must Cooperate in Better Nursing Relations.

Are We Making Progress in the Care of the Chronically Ill?

Hospitals and the Cancer Problem—Cancer Clinics and Cancer Detection Centers.

Newer Services for the Modern Hospital—Physical Medicine and Geriatrics.

Planned Convalescence and its Place in the Community.

Adjusting Hospital Service to Medical Progress.

Interdepartmental Relations of the Pharmacy; How to Enhance its Value and Appreciation by other Departments in the Hospital.

The Enlarging Educational Responsibilities of the Hospital.

Proper Use of the Medical Library.

Incentives to Hold Employees—Including Retirement Plans.

ROUND TABLE CONFERENCE—SPECIAL PROBLEMS OF THE SMALL HOSPITAL IN MEETING THE STANDARDS OF THE AMERICAN COLLEGE OF SURGEONS

Thursday, 2:00–5:00 p.m.—Sert Room—First Floor—The Waldorf-Astoria

Conducted by HARVEY AGNEW, M.D., Toronto; Secretary, Canadian Hospital Council; Editor of *Canadian Hospitals.*

Topics for Discussion:

Medical Staff Organization	Food Service
Clinical Laboratory	Pharmacy Service
X-ray Service	Personnel Management
Medical Records	Public Relations
Nursing Service	Care of Emergencies

Collaborators:

ALLAN D. CRAIG, M.D., New York; Hospital Consultant, Niergaard & Craig.

JANE S. DAVIS, M.H.A., Niles, Michigan; Superintendent, Pawating Hospital.

EVA H. ERICKSON, M.H.A.; Olean, New York; Superintendent, Olean General Hospital.

ELOISE B. FURNIVAL, M.H.A.; Syracuse; Administrative Assistant, Syracuse Memorial Hospital.

WILLIAM G. ILLINGER, White Plains, New York; Administrator, White Plains Hospital Association.

GEORGE PECK, Philadelphia; Administrator, Jewish Hospital.

MARSHALL I. PICKENS, Charlotte; Assistant Secretary, The Duke Endowment.

WILLIAM P. SLOVER, Manchester, Connecticut; Superintendent, Manchester Memorial Hospital.

ISABELLA N. WILLIAMS, Norwich, New York; Administrator, Chenango Memorial Hospital.

ROBERT G. WHITTON, Alexandria, Virginia, Administrator, Alexandria Hospital; Vice-President, The Hospital Council of the National Capital Area, Washington, D. C.

E. W. WILLIAMSON, M.D., Chicago; Assistant Director, American College of Surgeons.

SPECIAL CONFERENCE ON THE POINT RATING SYSTEM

Thursday, 8:00–10:00 p.m.—Sert Room—First Floor—The Waldorf-Astoria

A Special Conference on the Point Rating System for Hospitals with Particular Discussion of Evaluation and Rating of Hospitals under this System.

Conducted by: HENRY G. FARISH, M.H.A., M.D., Chicago; Assistant, Hospital Department, American College of Surgeons.

Collaborators:

HARVEY AGNEW, M.D., Toronto; Secretary, Canadian Hospital Council; Editor, *Canadian Hospitals.*

MALCOLM T. MACEACHERN, M.D., Chicago; Associate Director, American College of Surgeons.

PRELIMINARY CLINICAL PROGRAM

CLINICS IN NEW YORK HOSPITALS

Monday

BELLEVUE HOSPITAL

9:00–3:30. *Radiology.* Permanent Exhibit, Monday through Friday. Nonoperative Clinics. IRA I. KAPLAN and STAFF.

The Radiologic Treatment of Benign and Malignant Superficial Lesions. Irradiation as an Adjunct to Surgery. (I and K Bldg., Ground Fl.—Capacity 15).

The Place of Irradiation in the Treatment of Carcinoma of the Breast. (Same Rm.).

A New Approach to the Treatment of Gastro Intestinal Malignancies by Radiation. (Same Rm.).

Treatment of Carcinoma of the Cervix. (Same Rm.).

Radiation Therapy as an Adjunct Method of Treatment in Genito Urinary Neoplasm. (Same Rm.).

9:00–4:00. *Roentgenology.* Permanent Exhibit, Monday through Friday. Nonoperative Clinics: Roentgen Demonstrations of Surgical Disease.

Surgical Lesions of the Small Bowel Roentgenologically Considered. J. BUCKSTEIN. (X-Ray Dept. I and K Bldg., Ground Fl.—Capacity 15).

Surgical Problems of the Chest. PHILIP STRAX. (Same Rm.).

Diagnostic Problems in Osseus Disease. M. ZURROW. (Same Rm.).

Diagnosis of Renal Surgical Disease. H. ZUCKERMAN. (Same Rm.).

Interesting Neuro-Surgical Cases. LEWIS J. FRIEDMAN, HERBERT LOBSENS. (Same Rm.).

BELLEVUE HOSPITAL—FOURTH (NEW YORK UNIVERSITY) SURGICAL DIVISION

1:30–3:30. *Surgery, Specialties.* (Operating Rooms K-5 and K-6). (Capacity of each operating room, 5).

Children's Surgery—Hernia. PHILIP ALLEN (Operating Room A).

Children's Surgery—Chest. CHARLES W. LESTER (Operating Room B).

Maxillofacial Surgery—Surgical Correction of Prognathism of the Mandible. LEO WINTER (Operating Room C).

Orthopedic Surgical Clinic. ARTHUR KRIDA, JOHN MCCAULEY (Bone Room K-6).

BETH ISRAEL HOSPITAL

2:00–5:00. *Otorhinolaryngology.* Operative Clinic: Clinical Demonstration of Endoscopic Treatment. MAX SOM and STAFF. (E.N.T. O.R. 12th Fl.—Capacity 6).

Nonoperative Clinic on Results. MAX SOM and STAFF. (Auditorium—Capacity 200).

2:00–5:00. *Ophthalmology.* Nonoperative Clinic. EDWARD B. GRESSER and STAFF. (3rd Fl. Classroom—Capacity 6).

FLOWER AND FIFTH AVENUE HOSPITALS

2:00–4:00. *Neurosurgery.* Nonoperative Clinic: "Slipped Disc" Operation and End-Results. THOMAS I. HOEN. (Auditorium, 106th St. Entrance—Capacity 300).

4:00–5:00. *Thoracic Surgery.* Nonoperative Clinic: Trauma to Neck Involving the Mediastinum—or—Mediastinal Tumors. MILTON J. LLOYD. (Same Rm.).

HARLEM HOSPITAL

2:00–5:00. *Plastic Surgery.* Nonoperative Clinic: Symposium on Burns and Plastic Surgery. F. X. TIMONEY, J. A. TAMERIN, A. GARNES and STAFF. (Conference Rm. 5th Fl. Women's Pavilion—Capacity 100).

HOSPITAL FOR JOINT DISEASES

9:00–12:00. *Orthopedic Surgery.* Operative Clinic: Tendon Transplantation on the Hand. Transpositional Osteotomy at the Hip. LEO MAYER, HENRY MILCH. (Surg. Amphitheater—Capacity 50).

Follow-up Demonstration:

1. Result of Hallux Valgus Operation. PAUL LAPIDUS. (Prof. Serv. Bldg. Lecture Rm.—Capacity 60).
2. (a) Transplantation of Extensor Carpi Ulnaris Tendon to Give Abduction of the Thumb; (b) Meniscectomy of the Temporomandibular Joint; (c) Fracture of Xiphoid-Xiphoidectomy; (d) Osteotomy Fusion of Hip. MICHAEL BURMAN. (Same Rm.)
3. (a) Slipping of the Femoral Epiphysis and Results by the Use of Internal Rotation Brace; (b) Result of Osteotomy at the Hip Combined with Resection of Femoral Head. HENRY MILCH. (Same Rm.).
4. (a) Case Illustrating the Results of Tendon Transplantations and Nerve Suture; (b) Cases Illustrating Surgery of the Hand. LEO MAYER. (Same Rm.).

2:00–4:00. *Otorhinolaryngology.* Operative Clinic: Nose and Throat Surgery. L. KLEINFELD. (E.N.T. Surg. O.R.—Capacity 5).

LENOX HILL HOSPITAL

2:00–4:00. *Plastic Surgery.* Nonoperative Clinic: Demonstration of Cases. GUSTAV AUFRICHT. (Einhorn Auditorium—Capacity 450).

LYING-IN HOSPITAL

(See New York Hospital)

MANHATTAN EYE, EAR AND THROAT HOSPITAL

2:00–4:00. *Ophthalmology.* Nonoperative Clinic. Demonstration: Slit Lamp Demonstration. G. BONACCOLTO. (5th Fl. Exam. Rm.—Capacity 12).

Demonstration: Corneal Grafting Technique on Rabbits. H. M. KATZIN. (Same Rm.).

2:00–4:00. *Otolaryngology.* Demonstration: Biplane Fluoroscopy and Laminagraphy. W. R. CASHION. (X-Ray Lab. 6th Fl.—Capacity 24).

3:00–5:00. *Ophthalmology.* Demonstration: Cases of Cataract Surgery Complicated by Glaucoma. M. A. LAST. (Conference Rm. 2nd Fl.—Capacity 24).

MEMORIAL HOSPITAL

10:00–12:00. *Tumor Surgery.* Nonoperative Clinics: Sloan Kettering Institute. Cancer Research in Relation to Clinical Problems:

Methods of Chemotherapy and Some Clinical Applications. (A survey of methods used in the screening program and their application to patients with leukemia.) J. BURCHENAL. (Auditorium—Capacity 250).

Experiments on the Application of Chemotherapy Together with Conventional Methods. (Experimental

studies on the subject and examples of patients treated with mustards and x-rays). D. KARKOFSKY. (Same Rm.).

Cytochemical Studies of Mammary Cancer Including Observations During Hormone Therapy. (Presentation of methods and findings which would not include any of the data that Dr. Adair might use in his talk.) R. MELLORS. (Same Rm.).

Radioactive Iodine as a Tool in the Investigation and Treatment of Thyroid Cancers. F. FOOTE JR., MISS HULL. (Same Rm.).

Cases of Cancer of the Thyroid Treated with Radioactive Iodine. J. TRUNNEL, R. RAWSON. (Same Rm.).

METROPOLITAN HOSPITAL

2:00–4:00. *Neurosurgery.* Nonoperative Clinic: Peripheral Vascular Clinic Including Sympathectomy. LOUIS R. KAUFMAN, S. THOMAS GLASSER, ALBERT LESSER, KENNETH C. PEACOCK, DAVID SCHERF. (Conference Rm.—Capacity 60).

MONTEFIORE HOSPITAL

2:00–4:00. *General Surgery.* Nonoperative Clinic. SAMUEL STANDARD and STAFF. (Social Hall—Capacity 200).

4:00–5:00. *General Surgery.* Nonoperative Clinic: Neoplastic Diseases. DANIEL LASZLO. (Same Rm.).

MOUNT SINAI HOSPITAL

8:30–12:00. *Gynecology.* Operative and Nonoperative Clinics:

Parametrial Fixation Operation (Fothergill). Surgical Treatment of Urinary Stress Incontinence with Tantalum Plate. M. A. GOLDBERGER. (Surg. Amphitheater—Capacity 100).

Conservative Surgery for Pelvic Endometriosis. JOSEPH GAINES. (Same Rm.).

The Value of the Twenty-four Hour Pregnancy Test: a Review of 500 cases. EMANUEL KLEMPNER. (Same Rm.).

The Clinical Value of Hysterography. ARTHUR DAVIDS. (Same Rm.).

The Management of the Menopause. ROBERT I. WALTER. (Same Rm.).

2:00–5:00. *Neurosurgery.* Nonoperative Clinics:

Long Time Follow-up in Some Neurosurgical Cases. Case Presentations. ABRAHAM KAPLAN. (Surg. Amphitheater—Capacity 100).

Correlation of X-ray Findings and Clinical Picture. B. SCHLESINGER. (Same Rm.).

Dumbbell Tumor of the Spine. IRA COHEN. (Same Rm.).

Cranioplasty, Motion Picture Demonstration. S. GROSS. (Same Rm.).

NEW YORK EYE AND EAR INFIRMARY

2:00–3:30. *Otorhinolaryngology.* Nonoperative Clinic: Frontal and Ethmoid Sinusitis—Preoperative and Follow-up. STUART L. CRAIG. (Clinic Rm.—Capacity 20).

2:00–3:30. *Otorhinolaryngology.* Nonoperative Clinic: Antral Sinusitis—Preoperative and Follow-up. EARL F. LIMBACH. (Same Rm.).

4:00–6:00. *Otorhinolaryngology.* Operative Clinic: Sinus Surgery. STUART L. CRAIG. (Same Rm.).

4:00–6:00. *Otorhinolaryngology.* Operative Clinic: Surgery of the Antrum. EARL F. LIMBACH. (Same Rm.).

2:00–5:00. *Ophthalmology.* Nonoperative Clinic: Preoperative Diagnostic Study. CONRAD BERENS, H. ROMAINE and ASSOCIATES.

Preoperative and Postoperative Glaucoma Studies. W. KNIGHTON, H. ELWYN and ASSOCIATES.

Plastic Surgery Methods. W. HUGHES, J. COLE and ASSOCIATES. (Eye O.R. 10).

Special Lenses—Contact, Aniseikonic and Telescopic Exhibit. (Clinic Fl.—Capacity 30).

The routine clinics of the New York Eye and Ear Infirmary will be held as usual during this period; all Fellows of the American College of Surgeons are cordially invited to attend.

NEW YORK HOSPITAL

10:00–12:00. *Genitourinary Surgery.* Nonoperative Clinic: Demonstration: Surgical Anatomy. ERNEST LAMPE. (Bldg. A, Rm. 132—Capacity 15).

2:00–4:00. *General Surgery.* Nonoperative Clinic: Demonstration: Surgical Anatomy. Detailed Anatomy of Radical Neck Dissection. ERNEST LAMPE. (Same Rm.).

2:00–5:00. *Otolaryngology.* Operative Clinic: Reconstructive Surgery of the Nose. ARTHUR PALMER. (10th Fl. O.R.—Capacity 30).

NEW YORK HOSPITAL LYING-IN HOSPITAL

2:00–5:00. *Obstetrics and Gynecology.* Nonoperative Clinics:

Cryptomenorrhea—Congenital and Acquired. THOMAS L. BALL. (Lecture Room—Capacity 100).

Result of an Operation for Urinary Incontinence. ANDREW A. MARCHETTI, VICTOR MARSHALL. (Same Rm.).

Pregnancy Following Outlet Operation. WILLIAM FINN. (Same Rm.).

Studies on Patients with Hydramnious During the Past Fifteen Years. PAUL E. MUELLER. (Same Rm.).

End-Results of the Treatment of Carcinoma of the Endometrium. ANDREW A. MARCHETTI, JOHN E. MCALLISTER. (Same Rm.).

Radiological Survey of Breeches. H. L. WILCOX. (Same Rm.).

NEW YORK ORTHOPEDIC DISPENSARY AND HOSPITAL

8:00–12:00. *Orthopedic Surgery.* Operative Clinic. ALAN DE FOREST SMITH and STAFF. (O.R. 1 and 2—Capacity 16).

1:30–4:00. *Orthopedic Surgery.* General Orthopedic Clinic. HALFORD HALLOCK and STAFF. (7th Fl. Assembly Rm.—Capacity 30).

Congenital Club Foot Clinic. JOHN MCCAULEY. (Same Rm.).

NEW YORK POST-GRADUATE MEDICAL SCHOOL AND HOSPITAL

8:30–10:30. *Tumor Surgery.* Operative Clinic. H. W. MEYER. (O.R. 2—Capacity 10).

10:30–12:30. *Tumor Surgery.* Nonoperative Clinic: Treatment and Late Results in Carcinoma of the Head, Neck and Breast. H. W. MEYER and STAFF. (Erdmann Auditorium—Capacity 125).

2:00–5:00. *Vascular Surgery.* Operative Clinic. GERALD PRATT. (O.R. 1—Capacity 10).

2:00–5:00. *General Surgery.* Operative Clinic. J. W. HINTON. (Amphitheater—Capacity 50).

2:00–5:00. *Orthopedic Surgery.* Operative Clinic. G. ANOPOL. (O.R. 5—Capacity 10).

PRESBYTERIAN HOSPITAL

2:00–4:00. *General Surgery.* Nonoperative Clinic: Carcinoma of the Tongue at a General Hospital, Analyses of 100 cases. JOHN M. HANFORD. (McCosh Amphitheater, 18th Fl.—Capacity 142).

Lymphatic Spread of Carcinoma of the Rectum and Colon. ROBERT S. GRINNELL. (Same Rm.).
Roentgen Diagnosis of Carcinoma of the Stomach. ROSS GOLDEN. (Same Rm.).
Extent of Resection of Stomach for Carcinoma, Correlation with Experimental Mucus Transformation. Case Demonstration. EDWARD L. HOWES. (Same Rm.).
Chemotherapy of Malignancy with Nitrogen Mustard. ALFRED GELLHORN. (Same Rm.).
Surgical Anatomy: Demonstration of Autonomic Nerve Supply of the Gastrointestinal Tract. JOSE FERRER. (Rm. 514, 10th Fl., Phys. & Surg. Bldg.—Capacity 20).
Structures of the Female Perineum. PHILIP WIEDELL. (Same Rm.).

PRESBYTERIAN HOSPITAL
NEUROLOGICAL INSTITUTE

9:00–11:00. *Neurosurgery.* Nonoperative Clinic. Neurosurgical Staff Conference. Clinical preview of surgical cases to be operated upon Tuesday and Friday mornings. Surgical Staff. (Conference Rm., 2nd Fl.—Capacity 60).
Note: Regular operative clinics will be held every morning as usual and the visitors are welcome to attend.

PRESBYTERIAN HOSPITAL
SLOANE HOSPITAL FOR WOMEN

10:00–12:00. *Obstetrics and Gynecology.* Nonoperative and/or Operative Clinic: Demonstration of Delivery Room Technics and Teaching. Demonstration of Operative Obstetrics if Cases Available. CHARLES STEER, ANTHONY D'ESOPO, HOWARD C. MOLOY. (17th Fl. Clinic or O.R. or Lecture Rm.—Capacity 20).

PRESBYTERIAN-NEW YORK ORTHOPEDIC HOSPITALS Fracture Service

1:30–4:00 *Fractures and Traumatic Surgery.* Nonoperative Clinic: Fracture Clinic: The Low Back Problem. SAWNIE R. GASTON. (Rm. 417, 16th Fl., Phys. and Surg. Bldg.—Capacity 100).
Three, Four, and Five Year Hip Fracture Follow-up Results. CHARLES S. NEER. (Same Rm.).

ROOSEVELT HOSPITAL

2:00–4:00. *General Surgery.* Nonoperative Clinic: Discussion. Post-Graduate Education of the Surgical Residents and Interns. CHARLES F. STEWART. (Conference Rm. 1—Capacity 50).
2:30–5:00. *Fractures and Traumatic Surgery.* Nonoperative Clinic: Traumatic and Reconstructive Surgery of the Hand. CHARLES F. STEWART. (Conference Rm. 2—Capacity 50).
The Early Treatment of Hand Injuries (Emergency Treatment). CONDICT W. CUTLER. (Same Rm.).
The Repair of Finger Tip Injuries. HENRY A. KINGSBURY. (Same Rm.).
The Temporary Repair of Severed Flexor Tendons of the Fingers. R. STERLING MUELLER. (Same Rm.).
Reconstructive Surgery of the Hand. WILLIAM LITTLER.

ST. LUKE'S HOSPITAL

8:30–11:00. *General Surgery.* Operative Clinic. WILLIAM MACFEE and STAFF. (Lyle O.R.—Capacity 30).
11:00–1:00. *General Surgery.* Nonoperative Clinics: Clinical Demonstrations: Neurofibromas of Stomach, six cases. JOHN WEST. (Staff Conference Rm.—Capacity 60).
Management of Polyps of Colon and Rectum. PAUL MORTON. (Same Rm.).
Adenocarcinoma of Colon and Rectum with Metastasis to Ovaries, illustrative cases. WILLIAM BERRY. (Same Rm.).
Malignant Tumors of the Appendix. STAFFORD WEARN.
Bleeding Duodenal Ulcers. ROBERT FRASER. (Same Rm.).
Trends in Curare Technique of Anesthesia. EDWARD BURFORD. (Same Rm.).
2:00–5:00. *Orthopedic Surgery.* Operative Clinic. MATHER CLEVELAND and STAFF; DAVID BOSWORTH. (Lyle O.R.—Capacity 30).

ST. VINCENT'S HOSPITAL

9:00–12:00. *General Surgery.* Operative Clinic: General Surgery. RAYMOND P. SULLIVAN, JOHN A. LAWLER and GEORGE R. STUART, EDWARD DENNEEN. (Ward Bldg. O.R. A and B—Capacity 5 each; C, D and E—Capacity 10 each; F—Capacity 5).
9:00–12:00. *Thoracic Surgery.* Nonoperative Clinic: Group Conference: Selection of Cases for Thoracoplasty, Lobectomy and Pneumonectomy. DANIEL MULVIHILL. (Nurse's Lecture Rm. 2nd Fl.—Capacity 60).
9:00–12:00. *Radiology.* Nonoperative Clinic: Roentgen Diagnosis of Thoracic Tumors. WILLIAM W. MAYER and ASSOCIATES. (Ward Bldg. Viewing Rm.—Capacity 25).
10:00–12:00. *Neurosurgery.* Nonoperative Clinic: Follow-up Study of a Series of Herniated Discs with Fusion of Spine. JAMES T. DANIELS, ANTHONY PISANI. (Nurse's Auditorium—Capacity 150).
2:00–5:00. *Pathology.* Nonoperative Clinic: Clinical Pathological Demonstration of Hodgkin's Disease.
Growth of Tissue Anterior Chamber of the Eye of Mice. ANTHONY ROTTINO. (Nurse's Auditorium—Capacity 60).
Display and Demonstration of System Diseases by Kodachrome Slides. JOHN KEATING. (Same Rm.).
Display of Fixed Specimens. WILLIAM F. MEEHAN. (Same Rm.).

U. S. MARINE HOSPITAL
(STATEN ISLAND)

10:00–12:30. *General Surgery.* Nonoperative Clinic: Inguinal Hernia (Follow-up Study). Postoperative Incisional Hernia. HOMER L. SKINNER and STAFF. (Doctor's Staff Rm.—Capacity 75).
12:30–1:30. Luncheon at the Hospital.
1:00–3:00. *General Surgery.* Operative Clinic: Inguinal Hernia under Spinal or Continuous Caudal Anesthesia. HOMER L. SKINNER and STAFF. (General O.R., 4th Fl.—Capacity 20).
2:00–3:30. *Anesthesia.* Nonoperative Clinic: Diagnostic and Prognostic Regional Block Clinic. C. L. HEBERT and STAFF. (General O.R.—Capacity 5).

VETERANS ADMINISTRATION HOSPITAL

1:00–3:30. *General Surgery.* Nonoperative Clinic: General Surgical Conference. FREDERIC W. BANCROFT, FORDYCE B. ST. JOHN, ALLEN O. WHIPPLE, P. K. SAUER, CARNES WEEKS, JOHN E. SULLIVAN, EDWIN SELF, DAVID K. PINKS, MARTIN J. HEALY, and RESIDENT STAFF. (Bldg. A, 3rd Fl.—Capacity 200).
2:00–4:00. *General Surgery.* Operative Clinic: Endoscopy. DAVID H. JONES, HENRY THALER. (Bldg. B, 5th Fl.—Capacity 5).
3:30–5:00. *Radiology.* Nonoperative Clinic: Radiological Conference. CHARLES GOTTLIEB, ARCHIE SHEINMEL, MAXWELL H. POPPEL, and STAFF. (Bldg. A, 3rd Fl.—Capacity 200).

EXHIBITS
(Bldg. A, 3rd Fl.).

Anesthesia Section
Bronchogenic Carcinoma: Review of 600 Cases
Intracranial Neoplasms
Residency Training Program
Organization of Surgical Division
Results of Vagus Resection
Pathology Section
Medical Illustrations Laboratory
Plastic Surgery
Prosthetic Appliances

CLINICS IN BROOKLYN—LONG ISLAND HOSPITALS

Monday

CUMBERLAND HOSPITAL

2:00–5:00. *General Surgery*. Statistical Study. Discussion of Postoperative Complications. DAVID J. GRAUBARD (Auditorium—Capacity 300).

Use of Sulfonamides in Acute Abdomen. MICHAEL VENEZIANO. (Same Rm.).

Preoperative and Postoperative Treatment in Gastrointestinal Surgery. HARVEY COOPERMAN. (Same Rm.).

Advantage of Miller Abbott Tube in Small Bowel Obstruction. CESARO CASTELLANETA. (Same Rm.).

Late Results in Surgery of Gastrointestinal Malignancy with Pathological Demonstration. HAROLD AXELROD.

METHODIST HOSPITAL

2:00–4:00. *Plastic Surgery*. Operative and Nonoperative Clinic: Plastic Repair of Cleft Palate. JOHN F. FORD. (Main O.R. 1–6—Capacity 12).

2:00–5:00. *Otolaryngology*. Operative and Nonoperative Clinic: Otolaryngology. CHARLES A. ANDERSON, EINER A. SUNDE and STAFF. (Main O.R. 1–6—Capacity 50).

CLINICS IN NEW YORK HOSPITALS

Tuesday

BABIES HOSPITAL
(See Presbyterian Hospital)

BELLEVUE HOSPITAL—FIRST (COLUMBIA UNIVERSITY) SURGICAL DIVISION

8:00–10:00. *General Surgery*. Operative Clinic: FRANK B. BERRY and STAFF (O.R. A, B, C & D; O.R. K-5—Capacity 20).

8:00–12:00. *Thoracic Surgery*. Operative Clinic: FRANK B. BERRY and STAFF (O.R. C & D—Capacity 10).

10:30–12:30. *Nonoperative Clinic* (Stewart Amphitheatre I & K Bldg., Ground Fl.—Capacity 100).

Surgical methods in Treatment of Hypertension. CARNES WEEKS.

Chronic Thyroiditis. J. GORDON LEE.

Enterogenous Cysts. JAMES F. BAGG.

Treatment of Chronic Leg Ulcers by Fasciotomy. BERNARD J. RYAN.

Preliminary Report Concerning a Method for the Study of Peripheral Arterial Diseases. AARON HIMMELSTEIN.

BELLEVUE HOSPITAL—SECOND (CORNELL UNIVERSITY) SURGICAL DIVISION

2:00–5:00. *Neurosurgery*. Operative and Nonoperative Clinic. JOSEPH KING and WINFRID WINGEBACH; LAWRENCE POOL and HERBERT REIKERT. (O.R. K-5—Capacity 20).

BELLEVUE HOSPITAL—THIRD (NEW YORK UNIVERSITY) SURGICAL DIVISION

8:00–12:00. *Anesthesia*. Operative and Nonoperative Clinic. Demonstrations: Anesthesia in Thoracic Surgery. E. A. ROVENSTINE and STAFF. (5th Fl. C & D Bldg.—Capacity 5).

2:00–5:00 *General*. Rehabilitation in a General Hospital. Methods of Admission to Wards. GEORGE DEAVER, C. H. FUREY, Jr. (Wards F & G-3, F & G Bldg.—Capacity 25).

BETH ISRAEL HOSPITAL

9:00–12:00. *Plastic Surgery*. Operative Clinic: ARTHUR K. BARSKY. (O.R. A. 12th Fl.—Capacity 6).

2:00–5:00. *Genitourinary Surgery*. Operative Clinic. SEYMOUR WILHELM and STAFF. (Main O.R.—Capacity 36).

Nonoperative Clinic. SEYMOUR WILHELM and STAFF. (Auditorium—Capacity 200).

FLOWER AND FIFTH AVENUE HOSPITALS

9:00–11:00. *General Surgery*. Operative Clinic: Breast Surgery. HERBERT C. CHASE. (Main O.R. 8th Fl.—Capacity 12).

Nonoperative Clinic: Biological Studies in Malignancy. I. S. KLEINER, M. M. BLACK. (Auditorium—Capacity 300).

11:00–12:00. *General Surgery*. Nonoperative Clinic: Tumor Clinic. GEORGE T. PACK, JOHN O. VIETA, GEORGE K. HIGGINS, and FRANK J. BORELLI. (Same Rm.).

2:00–4:00. *Thoracic Surgery*. Nonoperative Clinic: Surgical Treatment of Angina Pectoris by the Establishment of Adhesive Pericarditis; Selection of the Patients; Operative Technique; Follow-up Results. SAMUEL A. THOMPSON, and M. J. RAISBECK. (Same Rm.).

4:00–5:00. *Obstetrics*. Nonoperative Clinic: Mannequin (Ayers Forceps, etc.) or—Ovarian Transplants. Two short moving pictures. BENJAMIN TOLY, HORACE AYERS. (Same Rm.).

GOLDWATER MEMORIAL HOSPITAL—SECOND SURGICAL DIVISION

9:30–10:30. *Geriatric Surgery*. Operative Clinics. CONDICT CUTLER and STAFF.

Adductor Tenotomy and Obturator Nerve Section for Adductor Spasm. (Adm. Bldg., Main O.R. 3rd Fl.—Capacity 30).

Repair of Scrotal Hernia; Local Anesthesia. (Same Rm.).

Amputation of Leg for Arteriosclerotic Gangrene; Refrigeration Anesthesia. (Same Rm.).

10:30–1:00. *Geriatric Surgery.* Nonoperative Clinic: Case Presentations:

Anesthesia Methods in Bad Risk Patients. E. A. ROVENSTINE. (North Conference Rm., 2nd Fl. Between C and D Pavilion—Capacity 50).

Clinical Studies in Chronic Peripheral Vascular Occlusion. GRANT P. PENNOYER. (Same Rm.).

Indications and Results of Hernia Repair in Old Patients. GEORGE STRENGER. (Same Rm.).

Problems in Chemical Balance in Geriatric Surgery. LOUIS CARP. (Same Rm.).

Dicoumarol and Other Anticoagulant Drugs. W. E. ALSOP. (Same Rm.).

Treatment of Chronic Bone Suppuration. F. B. ROTH. (Same Rm.).

Cholecystitis Complicating Chronic Disease States. W. T. MEDL. (Same Rm.).

Urgent Surgery in the Aged. CONDICT W. CUTLER, JR. (Same Rm.).

Clinical Pathological Demonstration. JULIUS ROSENTHAL. (Same Rm.).

2:00–4:00. *Geriatric Surgery.* Nonoperative Clinic: Case Presentations:

Arachnoiditis. IGNATZ OLJENICK. (B21 and 22—Capacity 50).

Clinical Evaluation of Cystometric Readings. J. E. BEGNER. (Same Rm.).

Preliminary Report on the Experimental Studies of Obstruction at the Vesical Neck Complicating the Neurogenic Bladder. ANDREW SPORER. (Same Rm.).

Vesical Neck Obstruction in the Female. LEA A. SHIFRIN. (Same Rm.).

Treatment of Incontinence in a Hospital for Chronic Diseases. SAMUEL SOIFER. (Same Rm.).

Suprapubic Prostatectomy, Modified Harris Technique with Complete Closure. S. A. RITTER. (Same Rm.).

Ward Rounds.

HARLEM HOSPITAL

9:00–12:00. *Traumatic Surgery.* Nonoperative Clinics: Trauma Symposium and Ward Rounds: Injuries of the Chest and Abdomen. R. H. YOUNG, J. C. WHITAKER, R. S. WILKINSON, M. LOGAN, F. R. ALLEN. (Wards 5th Fl. So. Women's Pavilion and 2A, 2B Main Bldg. and Conference Rm. 5th Fl. Women's Pavilion—Capacity 25).

2:00–3:00. *Genitourinary Surgery.* Nonoperative and Operative Clinic: Genitourinary Conference, Ward Rounds, and Operative Clinic. N. F. LASKEY, E. DONHEISER and STAFF. (Main Bldg. O.R. and Ward 2C—Capacity 25).

3:00–5:00. *Roentgenology.* Nonoperative Clinics: Roentgenology in Surgery:

Regional Ileitis—Intestinal Obstruction. L. GINSBURG. (Conference Rm. 5th Fl. Women's Pavilion—Capacity 100).

Duodenal Loop Triad. WILLIAM SNOW. (Same Rm.).

HOSPITAL FOR JOINT DISEASES

9:00–12:00. *Orthopedic Surgery.* Operative Clinic: Spine Fusion for Scoliosis. Saucerization for Chronic Osteomyelitis. SAMUEL KLEINBERG, JOSEPH BUCHMAN, THOMAS HORWITZ. (Surg. Amphitheater—Capacity 50).

Nonoperative Clinic: Miller Operation for Flat Feet. Use of Penicillin in the Operative Treatment of Chronic Osteomyelitis. The Operative Treatment of Cavus. Drilling of Incipient Epiphyseolisthesis at the Hip. SAMUEL KLEINBERG, JOSEPH BUCHMAN, THOMAS HORWITZ. (Lecture Rm.—Capacity 60).

2:00–4:00. *Orthopedic Surgery.* Nonoperative Clinic: Specimen Demonstration: The Pathology of Bones and Joints. HENRY JAFFE. (Same Rm.).

2:00–4:00. *Gynecology.* Operative Clinic: Total Hysterectomy for Uterine Tumors. Nonoperative Clinic: Follow-up Results of Total Hysterectomy for Uterine Tumors. HAROLD DAVIDSON. (Surg. Amphitheater—Capacity 50).

2:00–4:00. *Plastic Surgery.* Operative Clinic: Nasoplasty. Follow-up Clinic: Nasal, Lip and Face Plastics. LEOPOLD GLUSHAK. (Small O.R.—Capacity 5).

HOSPITAL FOR SPECIAL SURGERY

8:30–12:00. *General Surgery.* Operative Clinic: Operations by General Surgical Department. F. BEEKMAN and STAFF. (Main O.R.—Capacity 32).

9:00–12:00. *Orthopedic Surgery.* Nonoperative Clinic: Children's Orthopedic Problems. PHILIP D. WILSON, T. C. THOMPSON and STAFF. (Lecture Hall—Capacity 100).

2:00–5:00. *Orthopedic Surgery.* Nonoperative Clinic—Knee Problems:

Knee Capsuloplasty. LEWIS CLARK WAGNER. (Same Rm.).

Quadricepsplasty to Improve Knee Function. T. CAMPBELL THOMPSON. (Same Rm.).

Conservative Treatment of Fractures of the Tibial Plateau. ROBERT LEE PATTERSON. (Same Rm.).

Unusual Pathological Conditions Found in Knee Joints. MILTON HELPERN. (Same Rm.).

Old Pathological Fractures of Femora Treated with Knutschner Nail. JOHN R. COBB. (Same Rm.).

LENOX HILL HOSPITAL

9:00–11:00. *General Surgery.* Operative Clinic. FRANCIS M. DONEHUE and STAFF. (O.R. 1, 2 and 3—Capacity each 15).

11:00–12:00. *General Surgery.* Nonoperative Clinic: Therapeutic Considerations in Acute Small Bowel Obstruction. ROBERT C. CROWLEY. (Einhorn Auditorium—Capacity 450).

2:00–5:00. *Urology.* Nonoperative Clinics: Late Results of Unusual Cases:

Bilateral Stag Horn Renal Calculi. WILLIAM R. DELZELL. (Einhorn Auditorium—Capacity 450).

Stag Horn Calculus in Abnormally Rotated Ptosed Kidney. HERBERT KENYON. (Same Rm.).

Horseshoe Kidney Resection of Hydronephrotic Half. MAXIMILIAN NEMSER. (Same Rm.).

Operative Treatment of Large Dendritic Calculi of Solitary Kidney. GEORGE SLAUGHTER. (Same Rm.).

LYING-IN HOSPITAL

(See New York Hospital)

MANHATTAN EYE, EAR AND THROAT HOSPITAL

9:00–12:00. *Ophthalmology.* Operative Clinic: Surgical Treatment of Ptosis of Lids, Operation and Results. GUERNSEY FREY. (Frey Clinic, 1st Fl., and Eye O.R.—Capacity 5).

9:00–12:00. *Otolaryngology.* Operative Clinic. Nasal Plastic Operation. A. SCHATTNER. (O.R. 7th Fl.—Capacity 5).

10:00–12:00. *Otolaryngology.* Demonstration. Postoperative Cases of Laryngectomy; Esophageal Voice Training. D. S. CUNNING. (Broncho. Clinic, 3rd Fl.—Capacity 15).

2:00–4:00. *Ophthalmology.* Operative Clinic: Surgical Treatment of Strabismus, Operations and End-Results. F. C. KEIL. (Eye O.R., 6th Fl.—Capacity 5).
2:00–4:00. *Ophthalmology.* Orthoptic Clinic. F. C. KEIL, JR., E. KNAUBER. (Orth. Clinic, 1st Fl. Rear—Capacity 12).
2:00–5:00. *Otolaryngology.* Operative Clinic: Laryngectomy. D. S. CUNNING. (7th Fl. O.R. 1—Capacity 5).
2:30–5:00. *Otorhinolaryngology.* Operative Clinic: Nasal Plastic Operation. WILLIAM B. ALLAN. (7th Fl. O.R. 2—Capacity 5).
3:00–5:00. *Otolaryngology.* Clinicopathological Demonstration: Malignancy of the Larynx. A. A. EGGSTON, D. S. CUNNING. (Conference Rm. 2nd Fl.—Capacity 50).

MEMORIAL HOSPITAL

8:00–12:00. *Tumor Surgery.* Operative Clinics. FRANK E. ADAIR, G. E. BINKLEY, B. L. COLEY, A. L. DEAN, V. F. MARSHALL, HAYES MARTIN, GEORGE T. PACK, G. H. TWOMBLY, W. L. WATSON. (O.R. A—Capacity 15; O.R. B—Capacity 15; O.R. C—Capacity 7; O.R. D—Capacity 7; O.R. E—Capacity 7).
2:00–3:00. *Tumor Surgery.* Nonoperative Clinics: Discussion.
The Memorial Hospital Nomenclature for Cancer and Its Usefulness in Cancer Hospitals, Cancer Clinics, and Cancer Commissions. ELEANOR MACDONALD. (Auditorium—Capacity 250).
The Necessity for Uniform and Accurate Methods of Reporting Cancer Statistics; Cure Rates in Cancer Based Upon Total Experience. HAYES MARTIN. (Same Rm.).
The Organization of the Statistics Department of Memorial Hospital and a Report of Five-year End-Results in Various Anatomic Forms of Cancer as Calculated by the Statistics Department to Date. MARY MACDONALD. (Same Rm.).
3:00–4:00. *Tumor Surgery.* Nonoperative Clinic: Follow-up Patients With Total Laryngectomy for Cancer of the Larynx. (Same Rm.).
4:00–5:00. *Tumor Surgery.* Nonoperative Clinic: Follow-up Patients Illustrating Combined Neck Dissection and Excision of Primary Lesions of the Mouth. (Same Rm.).

METROPOLITAN HOSPITAL

9:00–12:00. *General Surgery.* Operative and Nonoperative Clinic: Thyroid Surgery and Resident Training. JAMES M. WINFIELD and STAFF. (Main O.R. and Conference Rm.—Capacity 60).
2:00–4:00. *Genitourinary Surgery.* Operative Clinic: Plastic Correction of Hydronephrosis; Ureterosigmoidostomy. SPRAGUE CARLETON and STAFF. (Main O.R. 1—Capacity 30; O.R. 2—Capacity 15; O.R. 3—Capacity 15).
Nonoperative Clinic: Demonstration: Selected Cases; Exhibit of Staff Training; Clinical Exhibit. SPRAGUE CARLETON and STAFF. (Same Rm.).

MONTEFIORE HOSPITAL

3:00–4:00. *General Surgery.* Nonoperative Clinic: Midleg Amputations in Diabetes. SAMUEL SILBERT. (Social Hall—Capacity 200).

MOUNT SINAI HOSPITAL

8:30–12:00. *Genitourinary Surgery.* Operative Clinic: Resection of the Rectum. Colectomy for Ulcerative Colitis. JOHN GARLOCK. (Surg. Amph.—Capacity 100).
Nonoperative Clinic and Case Presentations. JOHN GARLOCK, LEON GINZBURG, SAMUEL KLEIN, AMEIL GLASS, GABRIEL SELEY. (Same Rm.).
2:00–5:00. *Orthopedic Surgery.* Operative and Nonoperative Clinic. ROBERT LIPPMANN, ALBERT SCHEIN, EDGAR BICK. (Same Rm.).

NEUROLOGICAL INSTITUTE

(See Presbyterian Hospital)

NEW YORK EYE AND EAR INFIRMARY

10:00–12:00. *Otolaryngology.* Demonstration: Soundproof Room and Testing. J. SWIFT HANLEY. (Soundproof Rm. and Adjacent Rms.—Capacity 20).
2:00–3:00. *Otorhinolaryngology.* Demonstration: Frontal Sinus Surgery by Motion Pictures. J. SWIFT HANLEY. (Same Rm.).
4:00–6:00. *Otorhinolaryngology.* Nonoperative Clinic: Sinus Surgery. J. SWIFT HANLEY. (Weeks Hall—Capacity 100).
4:00–6:00. *Otolaryngology.* Nonoperative clinic: Radical Mastoidectomy. VIRGINIUS B. HIRST.
2:00–5:00.—*Ophthalmology.* Nonoperative Clinic.
Postoperative Results with Study of Operative Complications. BOYES and ASSOCIATES.
The Distribution of Newly Examined Patients and Outline for Further Studies. B. PAYNE and R. MCCLANAHAN and ASSOCIATES. (Clinic Rms.—Capacity 30).
Operative Clinic: Surgical Technique with Reference to Lids and Glaucoma. R. MEEK and W. JOHNSON and ASSOCIATES. (Eye O.R.—Capacity 10).
Orthoptic Department—Muscle Anomalies. (Orthoptic Clinic—Capacity 30).
The routine clinics of the New York Eye and Ear Infirmary will be held as usual during this period; all Fellows of the American College of Surgeons are cordially invited to attend.

NEW YORK HOSPITAL

9:00–12:00. *Thoracic Surgery.* Operative and Non-operative Clinics: FRANK GLENN and STAFF.
Surgery of Mediastinal Tumors. WILLIAM DEW. ANDRUS. (10th Fl. Amphitheater and Rm. F-639—Capacity 60).
Follow-up Studies of Mediastinal Tumors. JOHN ECKEL. (Same Rm.).
Chronic Constrictive Pericarditis. Surgical Aspects. WILLIAM DEW. ANDRUS. (Same Rm.).
Diagnosis of Carcinoma from Desquamated Cells. GEORGE PAPANICALAOU. (Same Rm.).
Clinical Value of Sputum Diagnosis. HENRY CROMWELL, WILLIAM A. BARNES. (Same Rm.).
9:00–12:00. *Genitourinary Surgery.* Operative and Non-operative Clinics: (From the Department of Surgery, New York Hospital—James Buchanan Brady Foundation).
Perineal Prostatectomy. THOMAS J. KIRWIN. (Surg. Amphitheater—Capacity 100).
Demonstration of Spinal Anesthesia. ROSE ANDRÉ. (Same Rm.).
Ureteropelvic Obstruction. ROY B. HENLINE. (Same Rm.).
Absorbable Gauze in Urologic Surgery. GEORGE FIEDLER. (Same Rm.).
Case Report. FRANCIS P. TWINEM. (Same Rm.).
True Adrenal Tumor of Kidney. ROBERT W. HUNT. (Same Rm.).
Uretral Obstruction in Relation to Psychosis. C. K. CHURCH. (Same Rm.).
Treatment of Bladder Tuberculosis with a New Instrument and Method. STANLEY WANG. (Same Rm.).

Glands in Female Urethra of the Infant. FRANCIS BENEVENTI. (Same Rm.).
Foreign Body in Ureter. ALBERT VERGES-PLAQUE. (Same Rm.).
Transplantation of Ureters. ALFONSO AVALOS. (Same Rm.).
Case Report. PAUL BOYD, ALBERTO GENTILE, WAYNE HANSON, CECIL HAWES. (Same Rm.).
Demonstration of Drawings of Interesting Urological Cases and Operations. WILLIAM P. DIDUSCH (Room F-956—Capacity 100).
Motion Pictures of Urological Operations. WILLIAM P. DIDUSCH (Room F-901—Capacity 100).
2:00–5:00. *General Surgery.* Nonoperative Clinic: General Surgery. FRANK GLENN and STAFF.
The Resident System in Surgical Training at the New York Hospital. FRANK GLENN. (Room B-001—Capacity 350).
Follow-up Results—Carcinoma of Breast, Stomach, Colon, Rectum. WILLIAM A. COOPER. (Same Rm.).
Organization of Follow-up Clinic. BRONSON S. RAY. (Same Rm.).
Demonstration: Follow-up Clinic Staff. (Same Rm.).
2:00–5:00. *Genitourinary Surgery.* Operative and Nonoperative Clinics:
The Treatment of Bladder Tumors (From the Department of Surgery, Cornell University Medical College, and Memorial and Bellevue Hospitals). VICTOR F. MARSHALL and STAFF.
Classification of Bladder Tumors. JOHN W. DRAPER. (Surg. Amphitheater—Capacity 100).
Results of Radiation Therapy of Bladder Tumor. VICTOR F. MARSHALL. (Same Rm.).
Transurethral Treatment of Bladder Tumors. ALLISTER M. MCLELLAN. (Same Rm.).
Bladder Resection for Bladder Tumor. J. EDWIN DREW. (Same Rm.).
Uretero-Cutaneous Transplantation. GUSTAVUS A. HUMPHREYS. (Same Rm.).
Ureterointestinal Anastomosis. MORRIS SCHNITTMAN. (Same Rm.).
Total Cystectomy. WILLET F. WHITMORE. (Same Rm.).
Management of Advanced Carcinoma of the Bladder. ROBERT S. HOTCHKISS. (Same Rm.).
Summary and Conclusions. VICTOR F. MARSHALL. (Same Rm.).
2:00–5:00. *Otolaryngology.* Operative Clinic: Surgery of the Mastoid. GERVAIS MCAULIFFE. (10th Fl. O.R. 1 Rm.—Capacity 30).
2:00–4:00. *General Surgery.* Nonoperative Clinic: Surgical Anatomy Demonstration: Anatomy of the Hand Involved in Infection and Trauma. ERNEST W. LAMPE. (Room A-132—Capacity 15).

NEW YORK HOSPITAL LYING-IN HOSPITAL

9:00–12:00. *Obstetrics and Gynecology.* Operative and Nonoperative Clinics:
Operation for Correction of Prolapse. BYRON GOFF. (Gyn. O.R., 2 Rms.—Capacity 50).
Extraperitoneal Cesarean Section. CURTIS MENDELSON. (Same Rm.).
Extraperitoneal Cesarean Section (moving picture) HERVEY WILLIAMSON. (Lecture Rm.—Capacity 100).
2:00–5:00. *Obstetrics and Gynecology.* Nonoperative Clinic:
Artificial Impregnation—Late Results. WILLIAM H. CAREY. (Same Rm.).
Clinic for Treatment and Study of Amenorrhea. RALPH W. GAUSE. (Same Rm.).
Cardiac Disease in Pregnancy. CURTIS L. MENDELSON. (Same Rm.).
Transfusion Reactions Due to Rh Incompatibility. ELMER KRAMER. (Same Rm.).
Obstetrical Experience in Rh-negative Women. JOHN T. Cole. (Same Rm.).
End-Results of Patients with Infertility. CHARLES M. MCLANE. (Same Rm.).

NEW YORK ORTHOPEDIC DISPENSARY AND HOSPITAL

9:00–12:00. *Orthopedic Surgery.* Rounds. End-Result Conference. ALAN DE FOREST SMITH. (7th Fl. Assembly Rm.—Capacity 30).
Low Back Pain and Herniation of the Nucleus Pulposus. ALAN DE FOREST SMITH. (Same Rm.).
Suppurative Arthritis. HALFORD HALLOCK. (Same Rm.).
1:30–4:00. *Orthopedic Surgery.* General Orthopedic Clinic. LEONIDAS A. LANTZOUNIS and STAFF. (Same Rm.).
Scoliosis Clinic. WILLIAM VON LOCKUM. (Same Rm.).

NEW YORK POST-GRADUATE MEDICAL SCHOOL AND HOSPITAL

8:30–11:30. *Urology.* Operative Clinic. CLARENCE BANDLER. (O.R. 5—Capacity 10).
8:30–12:00. *Plastic Surgery.* Operative Clinic. GUSTAVE AUFRICHT. (O.R. 2—Capacity 10).
10:30–12:30. *Neurosurgery.* Nonoperative Clinic: Case Presentations of Thoracolumbar Sympathectomy for Essential Hypertension. JERE W. LORD, JR., S. ARTHUR LOCALIO. (Erdmann Auditorium—Capacity 125).
11:30–1:00. *Oral Surgery.* Operative Clinic. F. S. DUNN. (O.R. 5—Capacity 10).
2:00–5:00. *Thoracic Surgery.* Operative Clinic. L. R. DAVIDSON. (O.R. 4—Capacity 10).
2:00–5:00. *General Surgery.* Operative Clinic. R. F. CARTER. (O.R. 5—Cap. 10).
2:00–5:00. *Gynecology.* Operative Clinic. W. T. DANNREUTHER. (Surg. Amphitheatre—Capacity 50).

PRESBYTERIAN HOSPITAL

8:00–12:00. *General Surgery.* Operative Clinics: Thoracic Surgery. GEORGE H. HUMPHREYS, HERBERT C. MAIER, RICHMOND L. MOORE. (Balcony Fl. 19, Main O.R. C–F—Capacity each 16).
Thyroid Surgery. WILLIAM B. PARSONS, LAWRENCE W. SLOAN, ROBERT H. E. ELLIOTT. (Same Rm.).
8:00–12:00. *Plastic Surgery.* Operative Clinics:
Plastic Operations. JEROME P. WEBSTER. (Balcony Fl. 19, Main O.R. G and H—Capacity each 16).
Release of Burn Contracture of the Neck. THOMAS W. STEVENSON. (Same Rm.).
2:00–4:00. *General Surgery.* Nonoperative Clinic: Surgical Treatment of Esophageal Neoplasm. GEORGE H. HUMPHREYS. (Amphitheater A, Main Fl., Phys. and Surg. Bldg.—Capacity 225).
The Surgical Treatment of Bronchiectasis. RICHMOND L. MOORE. (Same Rm.).
Mediastinal Tumors. HERBERT C. MAIER. (Same Rm.).
Thoracoabdominal Injuries. ROBERT H. WYLIE. (Same Rm.).
Surgery of Tumors of the Ampulla of Vater and Head of Pancreas. WILLIAM B. PARSONS. (Same Rm.).
Results of Partial Gastrectomy for Peptic Ulcer. HAROLD D. HARVEY. (Same Rm.).
2:00–4:00. *General Surgery.* Nonoperative Clinics: Experiences with Anticogulants in the Treatment of Thromboembolism. DAVID V. HABIF. (McCosh Amphitheater, 18th Fl.—Capacity 142).

Follow-up Results in Thyroid Operations. LAWRENCE W. SLOAN. (Same Rm.).

Physiological Studies in Injuries Due to Cold. OCTA C. LEIGH. (Same Rm.).

2:00–4:00. *Plastic Surgery.* Nonoperative Clinic:

Cases of Mammaplasty with Nipple and Areola Transplantation. Plastic Repair after Excision of Carcinoma. Plastic Repair after Extensive Burns. Plastic Repair in Miscellaneous Conditions. JEROME P. WEBSTER. (Rm. 417, 16th Fl. Phys. and Surg. Bldg.—Capacity 100).

Synovitis of the Wrist. Surgical Repair of X-Ray Burns. THOMAS W. STEVENSON. (Same Rm.).

PRESBYTERIAN HOSPITAL
BABIES HOSPITAL

9:00–11:00. *General Surgery.* Operative Clinic. EDWARD J. DONOVAN and STAFF. (O.R. 1 and 2, 9th Fl.—Capacity each 5).

11:00–12:00. *General Surgery.* Nonoperative Clinic: Presentation of Current Cases. (Ward Rounds—Capacity 10).

2:00–4:00. *General Surgery.* Nonoperative Clinic:

Congenital Hypertrophic Pyloric Stenosis, Report of 600 Consecutive Cases with Follow-up. EDWARD J. DONOVAN. (Amphitheater, 4th Fl.—Capacity 75).

Splenectomy in Children with Observations on Portocaval Anastomosis. LOUIS M. ROUSSELOT. (Same Rm.).

Duplications of the Alimentary Tract, Report of Seven Cases with Follow-up. THOMAS V. SANTULLI. (Same Rm.).

Intussusception. BRUCE M. HOGG. (Same Rm.).

PRESBYTERIAN HOSPITAL
NEUROLOGICAL INSTITUTE

9:00–12:00. *Neurosurgery.* Operative Clinic: Cases Previously Shown Clinically at the Monday Morning Staff Conference. Surgical Staff. (10th Fl. N.I.–O.R. 1—Capacity 12, O.R. 2—Capacity 6). Clinical Preview of Surgical Cases to be Operated Upon Wednesday, Thursday and Friday Mornings. Guests Welcomed.

12:30–2:00. Luncheon—Bard Hall.

2:00–4:30. *Neurosurgery.* Nonoperative Clinic: Summary Presentations of Current Research Projects. (2nd Fl. Conference Rm.—Capacity 60).

Experimental Production of Acute Disseminated Encephalomyelitis in Monkeys. ELVIN KABAT, ABNER WOLFF.

Experimental Studies of the Function of the Pyramidal System in Monkeys. FRED METTLER.

Localization of Level Lesions of the Spinal Cord by Electromyography. PAUL F. A. HOEFER.

Results of Treatment of Nonobstructive Hydrocephalus, Five-year Follow-up. JOHN E. SCARFF.

Premature Synostosis of the Sutures of the Cranial Vault —Treatment and Results. LESTER A. MOUNT.

Incidence of Convulsions Following Depressed Fractures of the Skull. A Follow-up Study. FRITZ CRAMER.

Surgical Procedures Directed to the Relief of Involuntary Mass Reflexes in Paraplegic Patients. J. LAWRENCE POOL.

Congenital Dermal Sinuses: Subdural Abscess; Abscess of Spinal Cord. LESTER A. MOUNT.

Anomalous Pacchionian Granulations as a Factor in Certain Types of Focal Epilepsy. Case Reports. JOHN E. SCARFF.

Therapeutic Applications of Curare Derivatives in Neurology—Motion Pictures. EDWARD B. SCHLESINGER.

4:30–5:00. Refreshments.

PRESBYTERIAN HOSPITAL
SLOANE HOSPITAL FOR WOMEN

10:00–12:00. *Obstetrics and Gynecology.* Nonoperative and/or Operative Clinic: Demonstration of Delivery Room Technics and Teaching. Demonstration of Operative Obstetrics if Cases Available. CHARLES STEER, ANTHONY D'ESOPO, HOWARD C. MOLOY. (17th Fl. Clinic or O.R. or Lecture Rm.—Capacity 20).

2:00–3:00. *Obstetrics and Gynecology.* Nonoperative Clinic: Late Results in Cesarean Section. ANTHONY D'ESOPO. (17th Fl. Lecture Rm.—Capacity 40).

3:00–4:00. *Obstetrics and Gynecology.* Nonoperative Clinic: Use of Stereoroentgenograms in the Diagnosis of Pelvic Types. HOWARD C. MOLOY. (Same Rm.).

4:00–5:00. *Obstetrics and Gynecology.* Nonoperative Clinic: Relation of Size of Fetal Head to Pelvic Inlet as Identified by Stereoroentgenograms. CHARLES STEER. (Same Rm.).

PRESBYTERIAN-NEW YORK ORTHOPEDIC HOSPITALS

Fracture Service

9:00–12:00. *Fractures and Traumatic Surgery.* Operative Clinic. FRACTURE SERVICE STAFF. (Presb. Hosp. Balcony 19th Fl. Main O.R.—Capacity 8).

2:00–4:00. *Orthopedic Surgery.* Nonoperative Clinic: Fracture Follow-up Clinic. FRACTURE SERVICE STAFF. (Rm. 237, 3rd Fl. Vanderbilt Clinic—Capacity 30).

4:00–5:00. *Orthopedic Surgery.* Nonoperative Clinic: X-Ray Review. FRACTURE SERVICE STAFF. (X-ray Rm. 25, Main Fl. Presb. Hosp.—Capacity 36).

ROOSEVELT HOSPITAL

2:00–4:00. *General Surgery.* Nonoperative Clinic: Surgical Treatment of Lesions of the Colon:

The Management of Polyps and Papillomas of the Rectum. FREDERICK H. AMENDOLA. (Conference Rm. 1—Capacity 50).

Pathology of Ulcerative Colitis. Gross and Microscopic Specimens (Illustrative Lantern Slides). WALTER W. BRANDES. (Same Rm.).

ST. LUKE'S HOSPITAL

8:30–11:00. *General Surgery.* Operative Clinic. WILLIAM MACFEE and STAFF. (Lyle O.R.—Capacity 30).

11:00–1:00. *General Surgery.* Nonoperative Clinic: Clinical Demonstrations: Unusual Arrest of Otherwise "Incurable Tumors." FRANCIS CARTER WOOD. (Staff Conference Rm.—Capacity 60).

Carcinoma of Breast, Ten-year Follow-up. BENJAMIN SHORE. (Same Rm.).

2:00–5:00. *Otolaryngology.* Operative Clinic: Operative Clinic and Clinical Demonstrations. WINSTON FOWLKES and STAFF. (Lyle O.R.—Capacity 6).

ST. VINCENT'S HOSPITAL

9:00–12:00. *General Surgery.* Operative Clinic: General Surgery. CONSTANTINE J. MAGUIRE, CLARENCE P. HOWLEY, LOUIS SANMAN. (O.R. A—Capacity 5).

9:00–12:00. *General Surgery.* Operative and Nonoperative Clinic: Surgery of the Large Bowel. FRANCIS X. TIMONEY, G. E. BINKLEY, M. R. DEDDISH. (O.R. C—Capacity 20).

9:00–12:00. *Pathology.* Nonoperative Clinic: Report on Study of the Question of Retrolymphatic Spread in Carcinoma of the Rectum. J. F. CONNELL. (O.R. D—Capacity 20).

9:00–12:00. *Genitourinary Surgery.* Nonoperative Clinic: Case Presentation: Diagnosis and Surgical Diseases of the Kidney. THOMAS F. HAWLEY, J. EDWIN DREW. (Nurse's Lecture Rm.—Capacity 60).

2:00–3:00. *Plastic Surgery.* Nonoperative Clinic: Round Table Discussion: Cleft Lip and Palate. Syndactylism. Skin Grafting of Burns. Postoperative Results and Presentation of Patients. WALTER A. COAKLEY, ROBERT H. CLIFFORD. (Same Rm.).

2:00–4:00. *Otolaryngology.* Operative Clinic and Demonstration: Oral and Laryngeal Malignancies and Non-Malignancies. JOHN M. LORE and ASSOCIATES. (O.R. D—Capacity 10).

2:00–4:00. *Pathology.* Nonoperative Clinic: Display and Demonstration: System Diseases by Kodachrome Slides. ANTHONY ROTTINO and ASSOCIATES. (Nurse's Home, Lab.—Capacity 30).

3:00–4:00. *Neurosurgery.* Nonoperative Clinic: Surgical Treatment of Tuberculoma of the Brain Supplemented with Streptomycin. T. Q. GARVEY, JR. (Nurse's Lecture Rm.—Capacity 60).

Morphology of the Oligodendroglia Demonstrated by a New Method. ANTONIO GRINO. (Same Rm.).

U. S. MARINE HOSPITAL (STATEN ISLAND)

10:00–12:30. *Orthopedic Surgery.* Nonoperative Clinics. A. A. MICHELE and STAFF.

Fracture of Olecranon. (Doctor's Staff Rm.—Capacity 75).

Fracture of Patella. Discussion and Presentation of Cases. (Same Rm.).

Treatment of Injuries; First Metacarpo-carpal Joint. (Same Rm.).

Trephine Vertebral Body. (Same Rm.).

10:00–12:30. *Thoracic Surgery.* Operative Clinics: First Stage Thoracoplasty Under Balanced Anesthesia. Pneumonectomy. H. L. SKINNER, R. K. IVERSON, L. R. DAVIDSON. (General O.R.—Capacity 20).

12:30–1:30. Luncheon at the Hospital.

1:00–3:00. *General Surgery.* Nonoperative Clinic: Surgical Ward Rounds. W. F. NICKEL and RESIDENT STAFF. (Surg. Wards, 3rd & 4th Fl.—Capacity 75).

1:00–3:00. *Orthopedic Surgery.* Operative Clinic: Iliac Graft; Non-union of Tibia. A. A. MICHELE and STAFF. (General O.R.—Capacity 10).

2:00–4:00. *Thoracic Surgery.* Nonoperative Clinics: Echinococcic Cyst. Discussion and Case Presentation. Chest Conference. R. K. IVERSON. (Doctor's Staff Rm.—Capacity 75).

2:30–4:00. *Anesthesia.* Nonoperative Clinic: Intravenous Procaine Therapy for Painful Orthopedic Condition. H. L. HEBERT and STAFF. (General O.R.—Capacity 5).

VETERANS ADMINISTRATION HOSPITAL

9:00–12:00. *General Surgery.* Operative Clinics. DAVID K. PINKS, EDWIN SELF, JOHN E. SULLIVAN. (Bldg. D, 2 Rms., 12th Fl.—Capacity 20).

9:00–12:00. *Genitourinary Surgery.* Operative Clinic. LOUIS SPIVACK, PERRIN SNYDER, WILLET F. WHITMORE. (Bldg. D, 1 Rm., 12th Fl.—Capacity 10).

9:00–12:00. *Plastic Surgery.* Operative Clinic. HERBERT CONWAY, ROBERT H. CLIFFORD. (Bldg. B, 1 Rm., 5th Fl.—Capacity 10).

9:00–12:00. *Radiology.* Radiotherapy of Head and Neck. RALPH E. HERENDEEN, BERNARD ROSWIT. (Bldg. C, Ground Fl.—Capacity 50).

9:00–12:00. *Otorhinolaryngology.* Operative Clinic. SYLVESTER DALY. (Bldg. B, 5th Fl.—Capacity 5).

9:30–11:30. *General Surgery.* Nonoperative Clinic: Medical Rehabilitation Clinic. HARRY KESSLER, KARL HARPUDER, IRWIN D. STEIN, BERNARD STOLL, JACOB BYER. (Bldgs. B-C-F, Ground Fl.—Capacity 20).

11:00–12:30. *General Surgery.* Nonoperative Clinic: Results of Operation for Carcinoma of the Rectum. DAVID K. PINKS. (Bldg. A, Ground Fl.—Capacity 200).

1:30–3:30. *General Surgery.* Nonoperative Clinic: Conference: Tumor Board. FRED STEWART, P. K. SAUER, ERNEST S. OLSON, BERNARD ROSWIT, BERNARD STRAUS, R. L. LACANNA, and RESIDENT STAFF. (Bldg. A, 3rd Fl.—Capacity 200).

3:30–5:00. *Pathology.* Nonoperative Clinic: Conference: Biopsies. FRED STEWART. (Same Rm.).

Sensitization to Rh-sub group. Case Report. ERNEST S. OLSON. (Same Rm.).

Results of Streptomycin in Tuberculosis of the Kidney. MORRIS RAKIETEN. (Same Rm.).

Hemoglobinuric Nephrosis. ARTHUR C. ALLEN. (Same Rm.).

Carcinoma of Islands of Langerhans Treated with Alloxan. BENJAMIN S. GORDON. (Same Rm.).

EXHIBITS
(Bldg. A, 3rd Fl.)

Anesthesia Section
Bronchogenic Carcinoma: Review of 600 Cases
Intracranial Neoplasms
Residency Training Program
Organization of Surgical Division
Results of Vagus Resection
Pathology Section
Medical Illustrations Laboratory
Plastic Surgery
Prosthetic Appliances

WOMAN'S HOSPITAL

8:30–12:30. *Obstetrics and Gynecology.* Nonoperative Clinics: ALBERT ALDRIDGE and STAFF.

Treatment of Neoplasms of the Female Genital Tract. Management of Birth Injuries and Uterine Displacements. (4th Fl. Conference Rm.—Capacity 75).

Diagnosis and Treatment of Obstetric Complications. End-Results of Treatment of These Conditions Will Be Discussed. (Same Rm.).

During the course of the morning the routine for training of Residents in Gynecology and Obstetrics will be outlined. (Same Rm.).

The pathologist will give demonstrations of obstetric and gynecologic pathological specimens. (Same Rm.).

CLINICS IN BROOKLYN—LONG ISLAND HOSPITALS

Tuesday

CUMBERLAND HOSPITAL

9:00–12:00. *Gynecology.* Operative Clinic: Gynecological Operations. SAMUEL LUBIN and STAFF. (O.R. 1 & 2—Capacity 20).

Preoperative and Postoperative Care of Gynecological Patients. CHARLES N. LOUGHRAN and STAFF. (Same Rm.).

10:30–11:00. *Gynecology.* Nonoperative Clinic: Granuloma Inguinale (Venereum) of Uterus—Follow-up. SILIK H. POLAYES. (Auditorium—Capacity 300).

2:00–4:30. *General Surgery.* Nonoperative Clinic: Iso-Immunization with A & B Factors in Erythroblastosis Fetalis. SILIK H. POLAYES. (Same Rm.).

Importance of Blood and Blood Derivatives in Obstetrics. LESLIE H. TISDALL. (Same Rm.).
Conservative Management of Abortions. RICHARD WALTMAN. (Same Rm.).
Bacillus Welchii Postpartum Infection, Late Results. CHARLES LOUGHRAN. (Same Rm.).

JEWISH HOSPITAL

8:00–10:00. *General Surgery.* Operative Clinics. LOUIS BERGER and STAFF. (Main O.R. Amphitheatre 1 & 2. Capacity each 25).
10:00–12:00. *General Surgery.* Nonoperative Clinic: Fascial Transplants in Repair of Large Incisional Hernia. LOUIS J. MORSE, JACOB S. BARB. (Auditorium—Capacity 200).
Bacillus welchii Infections of the Biliary Tract. (Same Rm.).
The Perforated Gall Bladder. BERNARD PINES. (Same Rm.).
Pitfalls in the Diagnosing and Surgical Therapy of Obstructive Jaundice. HYMAN L. BERSON, PAUL ASCHNER. (Same Rm.).
Experiences with Resection of the Vagus Nerves in the Treatment of Peptic Ulcer. THEODORE ARNETT. (Same Rm.).
Bowel Perforation with Miller Abbott Tube. SAMUEL ACHS. (Same Rm.).
Carcinoma of the Small Bowel. DAVID FARBER. (Same Rm.).
Delayed Versus Immediate Intestinal Anastomosis. LOUIS VERGER. (Same Rm.).
Surgical Indications in Chronic Ulcerative Colitis. RALPH WOLFE. (Same Rm.).

KINGS COUNTY HOSPITAL

8:30–1:00. *Obstetrics and Gynecology.* Operative Clinics (Long Island College Service). CHARLES A. GORDON, M. GLASS, W. S. MCGREGOR and F. P. LIGHT. (B. Bldg. O.R. 1–10—Capacity 15).
9:30–1:00. *General Surgery.* Operative Clinic: Operation for Carcinoma of the Colon. BENJAMIN CISSEL and STAFF. (Same Rm.).
9:30–1:00. *General Surgery.* Operative Clinic: Subdiaphragmatic Vagus Neurectomy. GEORGE G. DIXON and STAFF. (Same Rm.).
2:00–5:00. *General Surgery.* Nonoperative Clinic:
End-Result of Resection of the Head of the Pancreas for Carcinoma. BENJAMIN CISSEL and STAFF. (O.P.D. Bldg. Lecture Rm.—Capacity 100).
End-Result of Embolectomy of the External Iliac Artery. (Same Rm.).
Follow-up on Repair of Arteriovenous Fistula. BENJAMIN CISSEL and STAFF. (O.P.D. Bldg. Lecture Rm.—Capacity 100).
Follow-up on Unusual Results With Internal Fixation for Fracture of Femoral Neck. BENJAMIN CISSEL and STAFF. (Same Rm.).
2:00–5:00. *General Surgery.* Nonoperative Clinic:
Report on a Series of Vagus Neurectomies.
Gastroscopy and Gastric Surgery (RICHARD SCHWARTZ by request). (Main Bldg. Rm. B-3—Capacity 50).
Replacement Therapy by Massive Transfusions in Gastrointestinal Hemorrhage (VICTOR GINSBURG by request). (Same Rm.).
2:00–5:00. *Obstetrics and Gynecology.* Nonoperative Clinic: Gynecology. (Main Bldg. Lecture Rm., 1st Fl.—Capacity 40).
2:00–5:00. *Orthopedic Surgery.* Nonoperative Clinic: JOSEPH B. L'EPISCOPO and STAFF. (Main Bldg. Board Rm.—Capacity 50).
Follow-up on Hip Bone Blocks for Painful Hips in Adults. Presentation of Cases.
Secondary Joint Changes Following Burns. (Same Rm.).
Tuberculosis of the Greater Trochanter of the Femur, Follow-up Study. Complications. Presentation of Cases. (Same Rm.).
Abduction Hip Splint for Congenital Dislocation of the Hip Occipitocervicothoracic Fusion. Four-year Follow-up on an Unusual Case of Cervical Pott's Disease Complicated by Paraplegia. (Same Rm.).

LONG ISLAND COLLEGE HOSPITAL

9:00–12:00. *Obstetrics and Gynecology.* Operative and Nonoperative Clinics: Clinics and Demonstrations. Members of the Staff. (Surg. Amphitheater—Capacity 100).
2:00–4:00. *Genitourinary Surgery.* Operative Clinics: Operative Clinics, Demonstrations, Cases and End-Results. FEDOR SENGER and STAFF. (Same Rm.).

METHODIST HOSPITAL

8:00–12:00. *General Surgery.* Operative Clinic. HAROLD K. BELL, SEYMOUR G. CLARK and STAFF. (Main O.R. 1–6—Capacity 10).
8:00–12:00. *Orthopedic Surgery.* Operative Clinic. HENRY P. LANGE and STAFF. (Main O.R. Orthopedic Rm.—Capacity 5).
12:00–1:00. Luncheon at the Hospital.
2:00–5:00. *General Surgery.* Nonoperative Clinic: Symposium on Gastric and Duodenal Surgery. HENRY F. GRAHAM, Moderator.
The Peptic Ulcer Problem. H. K. BELL. (Nurse's Auditorium—Capacity 300).
The Value of X-Ray in Diagnosis of Peptic Ulcer. G. W. CRAMP. (Same Rm.)
The Value of the Gastroscope in Diagnosis of Peptic Ulcer. E. A. SUNDE. (Same Rm.).
Primary Carcinoma of the Duodenum. P. A. RENAUD. (Same Rm.).
Preoperative and Postoperative Care. M. HOEFLE. (Same Rm.).
Our Experience with Subtotal Gastrectomy. S. G. CLARK. (Same Rm.).
Vagotomy for Peptic Ulcer. E. H. GRIFFIN. (Same Rm.).
Report of Unusual Cases. J. A. TIMM. (Same Rm.).
Demonstration of Pathology. J. HOWE. (Same Rm.).
Demonstration of X-Rays. G. W. CRAMP. (Same Rm.).

CLINICS IN NEW YORK HOSPITALS

Wednesday

BABIES HOSPITAL

(See Presbyterian Hospital)

BEEKMAN-DOWNTOWN HOSPITAL

9:30–10:45. *Traumatic Surgery.* Nonoperative Clinic: Ward Fracture Rounds. ROBERT H. KENNEDY and STAFF. (Wards. Total Capacity 30. In groups 5–6).
10:45–12:00. *Traumatic Surgery.* Nonoperative Clinics: Sensory Denervation of the Heel for Persistent Pain Due to Os Calcis Fracture. MYRON A. SALLICK. (3rd Fl. Annex. Capacity 30).
Indications for Myotomy in Flexor Tendon Injuries. LESTER BLUM. (Same Rm.).

Abdominal Puncture as a Diagnostic Aid in the Acute Abdomen. SIGMUND MAGE. (Same Rm.).
General Discussion of Patients Seen on Rounds. ROBERT H. KENNEDY and STAFF. (Same Rm.).

BELLEVUE HOSPITAL—FIRST (COLUMBIA UNIVERSITY) SURGICAL DIVISION

8:00–12:00. *Thoracic Surgery.* Operative Clinic. FRANK B. BERRY and STAFF. (O. R. C & D, 5th Fl.—Capacity 10).

BELLEVUE HOSPITAL—SECOND (CORNELL UNIVERSITY) SURGICAL DIVISION

8:30–10:30. *General Surgery.* Operative Clinics. GUILFORD S. DUDLEY and STAFF. (K-5 O.R.—Capacity 5).
Abdominoperineal Resection for Carcinoma of Rectosigmoid. GUILFORD S. DUDLEY (O.R. A).
Split Thickness Skin Graft to Arm. CARLETON CORNELL (O.R. B—Capacity 5).
Subtotal Gastrectomy for Peptic Ulcer. CRANSTON HOLMAN (O.R. C—Capacity 5).
Thoracolumbar Sympathectomy. ERNEST W. LAMPE (O.R. D—Capacity 5).
10:30–1:00. *General Surgery.* Nonoperative Clinics (C & D Amph.—Capacity 100).
Report on a Series of Esophagogastrostomy Cases. LAURENCE MISCALL.
Report on 17 Cases of Richter's Hernia. DAVID LYALL, RAYMOND LUOMANEN.
Report on 120 Cases of Internal Fixation of Fractures of Neck of Femur. RAYMOND LUOMANEN, JOHN PRUDDEN.
Split Thickness Grafts in General Surgery. CARLETON CORNELL.
Report on a Series of Peptic Ulcer Perforations. S. W. MOORE, ROBERT HENDRICHS.
Report on a Series of 7 Cases of Gastrojejunocolic Fistula. HERBERT REIKERT.
2:00–4:30. *Genitourinary Surgery.* Operative and Nonoperative Clinic; ROBERT HOTCHKISS and HOWARD JECK; G. W. DRAPER and GEORGE SLAUGHTER; GUSTAV HUMPHREYS and JOSEPH REIKERT; DEAN MAKOWSKI. (K-6 O.R. Capacity 5).

BELLEVUE HOSPITAL—THIRD (NEW YORK UNIVERSITY) SURGICAL DIVISION

8:00–12:00. *Anesthesia.* Operative Clinic. Anesthesia in Thoracic Surgery. E. A. ROVENSTINE and STAFF. (8th Fl. C & D O.R.—Capacity 5).
2:00–5:00. *General.* Nonoperative Clinic. Demonstration: Rehabilitation and Physical Medicine for Hemiplegias, Rehabilitation Programs for Hemiplegic Patients. GEORGE DEAVER. (Wards F & G-3, 3rd Fl. F & G Bldg.—Capacity 25).

BETH ISRAEL HOSPITAL

9:00–12:00. *Neurosurgery.* Operative Clinic: SIDNEY GROSS and STAFF. (Neurosurgical O.R. 12th Fl.—Capacity 6).
2:00–5:00. *General Surgery.* Operative Clinic: LEON GINZBURG, SAMUEL STANDARD, and STAFF. (Main O.R., 12th Fl.—Capacity 36).
2:00–5:00. *Bacteriology and Serology.* Nonoperative Clinic: Penicillin and Streptomycin. ERICH SELIGMANN. (3rd Fl., Lecture Rm.—Capacity 30).

FLOWER AND FIFTH AVENUE HOSPITALS

9:00–10:00. *General Surgery.* Operative Clinic: A New Operation for Pilonidal Cyst. JOHN HERRLIN. (Main O.R. 8th Fl.—Capacity 12).
Nonoperative Clinic: Preoperative and Postoperative Care. LOUIS R. KAUFMAN, JAMES M. WINFIELD and STAFF. (Auditorium—Capacity 300).
10:00–12:00. *Genera Surgery.* Nonoperative Clinic: Test for Viability of Bowel. JOHN HERRLIN, KURT LANGE, S. THOMAS GLASSER.
Nonoperative Clinic: Intestinal Obstruction. ROBERT T. CROWLEY. (Auditorium—Capacity 300).
2:00–4:00. *Orthopedic Surgery.* Nonoperative Clinic: Orthopedic Surgery. ANSON H. BINGHAM. (Same Rm.).
2:00–4:00. *Fractures.* Nonoperative Clinic: Fracture Treatment—Motion Pictures and End-Results—Demonstrations. MILTON J. WILSON and STAFF. (Same Rm.).
2:00–4:00. *Plastic Surgery.* Operative Clinic: Plastic Surgery (Straatsma). DAVID M. MAYER, CLARENCE R. STRAATSMA, JOHN F. FORD. (Main O.R. 8th Fl.—Capacity 12).
Nonoperative Clinic: End Results. DAVID M. MAYER, CLARENCE R. STRAATSMA, JOHN F. FORD. (Nurse's Lecture Rm. 8th Fl.—Capacity 25).
2:00–4:00. *Urology.* Nonoperative Clinic: Teaching Exhibit in Outpatient Department. SPRAGUE CARLETON and STAFF. (O.P.D. 105th St. Entrance—Capacity 25).
Nonoperative Clinic: McGregor Orchidectomy. L. P. WERSHUB. (Room A-41—Capacity 18).

GOLDWATER MEMORIAL HOSPITAL—THIRD SURGICAL DIVISION

9:00–10:00. *Geriatric Surgery.* Operative Clinic. HOWARD BARBER and STAFF. (Adm. Bldg. Main O.R. 3rd Fl.—Capacity 30).
9:00–11:30. *Geriatric Surgery.* Nonoperative Clinics:
Refrigeration in Clinical Surgery. W. F. RUGGIERO.
Primary Closure in Amputations of Lower Extremity. M. J. TOBIAS.
Herniorrhaphy in the Aged. M. STANLEY-BROWN.
Arteriectomy. NED SCHNAYERSON.
Peritonitis in the Aged. T. C. CASE.
11:30–12:00. *Geriatric Surgery.* Nonoperative Clinic: Ward Rounds. (C 21 and 22).
1:00–4:30. *Geriatric Surgery.* Nonoperative Clinics:
The Treatment of Varicose Ulcers by Ligation of Femoral Vein. LEO KENNEDY. (North Conference Rm. 2nd Fl. Between C and D Pavilion—Capacity 50).
Pancreatitis in the Aged. HENRY DOUBILET. (Same Rm.).
Prostatectomy in the Complicated Case. SAMUEL S. BEIRSTEIN. (Same Rm.).
Interesting Urological Conditions in the Aged. GEORGE SLAUGHTER. (Same Rm.).
Intravenous Procaine in Surgery. MILTON OPPENHEIM. (Same Rm.).
Arteriography. EUGENE TORTORA. (Same Rm.).
Combined Lumbar Sympathectomy and Vein Ligation for Peripheral Vascular Disease. B. SHAFIROFF. (Same Rm.).
4:30–5:30. *Geriatric Surgery.* Nonoperative Clinic: Forum on Surgical Investigation by Postgraduate Class of Surgery. (Same Rm.).

HARLEM EYE AND EAR HOSPITAL

8:00–9:00. *Otolaryngology.* Operative Clinic: Tonsillectomies with Meding tonsil enucleator. C. B. MEDING, E. GRABSCHEID and STAFF. (Tonsil O.R.—Capacity 5).
9:00–10:00. *Ophthalmology and Otorhinolaryngology.* Operative Clinic: Major Eye, Ear, Nose and Throat Surgery. A. HEATHERINGTON. (Major O.R.—Capacity 5).
10:00–11:00. *Otorhinolaryngology.* Demonstration: Demonstration of Ear, Nose and Throat Cases. C. B. MEDING and STAFF. (E.N.T. Clinic—Capacity 20).

11:00–12:00. *Otorhinolaryngology.* Operative Clinic: Rhinoplastic Surgical Case. H. SAFIAN. (Major O.R.—Capacity 5).

12:00–2:00. *Ophthalmology.* Operative Clinic: Eye Surgery. C. B. MEDING, P. MULLER. (Major O.R.—Capacity 5).

HARLEM HOSPITAL

9:00–12:00. *General Surgery.* Nonoperative Clinic: Symposium on Lymphogranuloma and Rectal Strictures. Ward Rounds. L. T. WRIGHT, J. G. LEVY, and STAFF. (Ward 5 W. Women's Pavilion—Capacity 25).

2:00–5:00. *Gynecology.* Symposium. H. C. FALK, P. M. MURRAY, and STAFF.

Operative Clinic in Gynecological Surgery. (Gyn. O.R. 4th Fl. Women's Pavilion—Capacity 8).

Nonoperative Clinics: (a) Management of Postabortal Infection; (b) Management of Recurrent Salpingitis; (c) Management of Pyosalpinx Complicating Fibroid with End-Results. (Conference Rm. 4th Fl. Women's Pavilion—Capacity 8).

HOSPITAL FOR JOINT DISEASES

9:00–10:30. *Orthopedic Surgery.* Operative Clinic: Nerve Resection for Painful Arthritis of Hip Joint. ISADORE ZADEK. (Surg. Amphitheater—Capacity 50).

9:30–12:00. *Genitourinary Surgery.* Operative Clinic: Hemorrhoids. Fistula-in-ano. Benign Adenoma of Rectum. Pilonidal Cyst. Nonoperative Clinic: Lymphogranuloma Venereum. HARRY GOLDMAN. (Small O.R.—Capacity 5).

2:00–4:00. *Genitourinary Surgery.* Operative Clinic: Surgery of the Kidneys, Ureter and Bladder. PAUL ASCHNER. (Surg. Amphitheater—Capacity 50).

HOSPITAL FOR SPECIAL SURGERY

8:30–12:00. *Orthopedic Surgery.* Operative Clinic. Children's Service. PHILIP D. WILSON and STAFF. (Main O.R.—Capacity 32).

9:00–12:00. *Orthopedic Surgery.* Nonoperative Clinic: General Surgical Problems. F. BEEKMAN, R. V. GRACE, B. L. COLEY, and STAFF. (Lecture Hall—Capacity 100).

2:00–5:00. *General and Plastic Surgery.* Nonoperative Clinic: General Surgical Problems and Plastic Surgery. F. BEEKMAN and STAFF. (Same Rm.).

LENOX HILL HOSPITAL

9:00–12:00. *General Surgery.* Operative Clinic. OTTO C. PICKHARDT and STAFF. (O.R. 1, 2 and 3—Capacity each 15).

11:00–12:00. *General Surgery.* Nonoperative Clinic: Experiences in Vagotomy for Peptic Ulcer. JAMES R. DONALDSON. (Einhorn Auditorium—Capacity 450).

2:00–4:00. *Thoracic Surgery.* Nonoperative Clinic: Late Results: Following Pneumonectomy and Lobectomy in Bronchiectasis, Cystic Disease of the Lung and Polmonary Abscess. HERBERT C. MAIER, WALTER FISCHER. (Same Rm.).

LYING-IN HOSPITAL
(See New York Hospital)

MANHATTAN EYE, EAR AND THROAT HOSPITAL

9:00–12:00. *Ophthalmology.* Nonoperative Clinic. Demonstration of Aniseikonia Clinic. A. LINKSZ. (1st Fl. Rear—Capacity 12).

9:00–12:00. *Ophthalmology.* Operative Clinic: Plastic Operations of Eye. BYRON SMITH. (Eye O.R. 6th Fl.—Capacity 5).

9:00–12:00. *Otolaryngology.* Fenestration Demonstration: Postoperative Cases. M. F. JONES, R. J. BELLUCCI. (Jones Clinic 2nd Fl.—Capacity 24).

9:30–12:00. *Ophthalmology.* Demonstration, Glaucoma Clinic: Late Results of Glaucoma Surgery. F. L. P. KOCH. (East Eye Clinic 1st Fl.—Capacity 24).

10:00–12:00. *Ophthalmology.* Demonstration: Corneal Grafting Technique on Rabbits. H. M. KATZIN. (Eye Bank Lab. 10th Fl.—Capacity 12).

10:00–12:00. *Otorhinolaryngology.* Operative Clinic and Demonstration: Operation for Polypoid Sinusitis and Demonstration of Postoperative Cases. A. NIGRO. (7th Fl. O.R.—Capacity 5).

1:00–3:00. *Ophthalmology.* Demonstration: Corneal Grafting Technique: Corneal Research Laboratory. R. T. PATON, H. M. KATZIN. (Eye Bank Lab. 10th Fl.—Capacity 12).

1:30–4:00. *Otolaryngology.* Bronchoscopic Clinic: Diagnosis and Treatment. D. S. CUNNING. (Broncho. Clinic 3rd Fl.—Capacity 12).

2:00–5:00. *Otolaryngology.* Operative Clinic: Fenestration Operation. M. F. JONES. (O.R. 6th Fl.—Capacity 2).

2:00–4:00. *Ophthalmology and Otorhinolaryngology.* Demonstration: Allergy in Ophthalmology. Allergy in Otorhinolaryngology; Demonstration of Cases and Testing Technique. A. A. EGGSTON, A. B. PAUL. (Conference Rm.—Capacity 50).

2:00–4:00. *Otolaryngology.* Operative Clinic: Tonsil Surgery. WILLIAM H. TURNLEY. (O.R. 7th Fl.—Capacity 5).

2:00–4:00. *Ophthalmology.* Operative Clinic: Cataract Surgery. D. H. WEBSTER. (Eye O.R. 6th Fl.—Capacity 5).

3:00–5:00. *Ophthalmology.* Demonstration: Eye Pathology: Demonstration Microscopic Sections. JOSEPH LAVAL. (Eye Path. 2nd Fl.—Capacity 50).

MEMORIAL HOSPITAL

8:00–12:00. *Tumor Surgery.* Operative Clinics. FRANK E. ADAIR, G. E. BINKLEY, B. L. COLEY, A. L. DEAN, V. F. MARSHALL, HAYES MARTIN, GEORGE T. PACK, G. H. TWOMBLY, W. L. WATSON. (O.R. A—Capacity 15; O.R. B—Capacity 15; O.R. C—Capacity 7; O.R. D—Capacity 7; O.R. E—Capacity 7).

10:00–12:00. *Tumor Surgery.* Nonoperative Clinics: Sloan Kettering Institute. Biochemical Changes in Patients with Cancer:

Altered Steroid Excretion of Patients with Cancer and Other Diseases. K. DOBRINER, S. LIEBERMANN. (Auditorium—Capacity 250).

Altered Carbohydrate Metabolism in Patients with Gastric Cancer. N. F. YOUNG, F. HOMBURGER. (Same Rm.).

The Interpretation of Protein Determinations. M. L. PETERMANN, N. F. YOUNG, K. HOGNESS. (Same Rm.).

Altered Protein Metabolism and the Clinical Implications of the Systemic Disorder in Patients with Gastric Cancer. F. HOMBURGER, N. F. YOUNG, E. C. REIFENSTEIN, JR. (Same Rm.).

The Modern Orientation of Clinical Cancer Research, a Discussion of the Preceding Papers. C. P. RHOADS. (Same Rm.).

Revised Hayes Martin Clinic. (Same Rm.).

Castration in Carcinoma of Male Breast. N. E. TREVES. (Same Rm.).

2:00–3:00. *Tumor Surgery.* Nonoperative Clinic: Follow-up Clinics in Cancer of the Uterus. G. H. TWOMBLY. (Auditorium—Capacity 250).

3:00–4:00. *Tumor Surgery.* Nonoperative Clinic: The Conduct of End-Results in Early Cancer Detection Clinic. ELISE L'ESPERANCE. (Same Rm.).

METROPOLITAN HOSPITAL

9:00–10:00. *General Surgery.* Non-Operative Clinic: Intestinal Obstruction. CHARLES A. HALBERSTAM, ROBERT T. CROWLEY. (Conference Rm.—Capacity 60).

10:00–12:00. *Genitourinary Surgery.* Operative Clinic: Colon Surgery. HAROLD E. CLARK. (Same Rm.).
Intestinal Intubation. WALTER L. MERSHEIMER. (Same Rm.).

2:00–4:00. *Thoracic Surgery.* Operative Clinic: Value of Cavernostomy in Tuberculosis (2 Operations). SAMUEL A. THOMPSON and STAFF. (Main O.R. 1—Capacity 30).

2:00–4:00. *Neurosurgery.* Operative Clinic: Nerve Suture; Cases. THOMAS I. HOEN, IGNAZ OLJENICK and STAFF. (Main O.R. 2—Capacity 15).

MONTEFIORE HOSPITAL

10:00–12:00. *General Surgery.* Nonoperative Clinic: Results of Treatment of Hydrocephalus by Choroid Plexectomy. LEO DAVIDOFF. (Social Hall—Capacity 200).

2:00–5:00. *General Surgery.* Operative Clinic. LEO DAVIDOFF. (O.R.—Capacity 10).

3:00–5:00. *Ophthalmology.* Ophthalmological Service:
Vitreous Transplantation. S. GARTNER, B. PRIESTLEY. (Social Hall—Capacity 200).
Visual Field Studies in Neurosurgery. M. CHAMLIN. (Same Rm.).
Electroretinography. E. BILLET.
Modified Tarsorrhaphy for High Degrees of Exophthalmos. S. GARTNER, M. CHAMLIN.
Cataract Surgery in Young Diabetics. A. I. LANCHNER.

MOUNT SINAI HOSPITAL

8:30–10:00. *Thoracic Surgery.* Operative Clinic: Operation for Patent Ductus Arteriosus. ARTHUR S. W. TOUROFF. (Surg. Amph.—Capacity 100).

10:00–12:00. *Thoracic Surgery.* Nonoperative Clinic:
Intramuscular Heparin (Concentrated) Therapy in the Prevention of Post-Operative Thrombosis and Embolism. HAROLD NEUHOF. (Surg. Amph.—Capacity 100).
Technique of Operation for Clotted Hemothorax (Motion Picture Demonstration). (Same Rm.).
Results of Operative Treatment of Traumatic Thoracic Lesions in Civilian Practice. ARTHUR S. W. TOUROFF. (Same Rm.).
Operative Results in Pediatric Surgery. ERNEST E. ARNHEIM. (Same Rm.).
Results of Surgical Treatment of Perforation of the Esophagus. EDWARD A. JEMERIN. (Same Rm.).
Results of Conservative Surgical Treatment of Chronic Pulmonary Abscess. IRVING A. SAROT. (Same Rm.).
Late Results in Pulmonary Resection for Carcinoma of the Lung. (Same Rm.).

2:00–5:00. *Ophthalmology.* The Surgical Treatment of Glaucoma. JOSEPH LAVAL. (Same Rm.).
Operative and Nonoperative Clinic. Operated Cases. HENRY MINSKY, JOSEPH LAVAL, DAVID WEXLER. (Same Rm.).

NEW YORK EYE AND EAR INFIRMARY

10:00–12:00. *Otolaryngology.* Nonoperative Clinic: Fenestration—Preoperative and Follow-up. J. M. SMITH. (Clinic Rm.—Capacity 20).
Otolaryngology. Plastic Surgery-Preoperative and Follow-up. JAY D. WHITHAM. (Same Rm.)

2:00–5:00. *Otolaryngology.* Operative Clinic: Fenestration Operation. J. M. SMITH. (Ear O. R.—Capacity 5.)
Otolaryngology. Operative Clinic: Plastic Surgery. JAY D. WHITHAN. (Same Rm.)

2:00–5:00. *Ophthalmology.* Nonoperative Clinic.
Indications for Surgical Interference. W. KNIGHTON and H. ELWYN and ASSOCIATES. (Clinic Rms.—Capacity 30.)
The Follow-up on Plastic Surgery with Discussion of Further Steps. W. HUGHES, J. COLE and ASSOCIATES.
Operative Clinic: Surgical Demonstration with Emphasis on Cataract and Glaucoma Surgery. C. BERENS and H. ROMAINE and ASSOCIATES. (Eye O. R.—Capacity 10).
X-Rays and Localization. IRVING SCHWARTZ.
The routine clinics of the New York Eye and Ear Infirmary will be held as usual during this period; all Fellows of the American College of Surgeons are cordially invited to attend.

NEW YORK HOSPITAL

9:00–11:30. *Neurosurgery.* Nonoperative Clinic: Symposium: Conducted by BRONSON S. RAY.
Surgical Relief of Intractable Pain of the Head and Neck. HERBERT PARSONS. (Room F-639—Capacity 70).
Oscillographic Study of Sensory Pathways: Evidence of Bilateral Conduction. CHARLES BERRY, RICHARD KARL, JOSEPH C. HINSEY. (Same Rm.).
Alterations in Visceral Sensation After Thoracolumbar Sympathectomy. CHARLES L. NEILL. (Same Rm.).
Induced Mechanical Stresses in the Analysis of Headache Mechanisms and the Diagnosis of Intracranial Tumors. E. CHARLES KUNKLE. (Same Rm.).
Sympathetic Supply to the Lower Extremities. ARTHUR D. CONSOLE. (Same Rm.).
On the Nature of Hyperalgesia. HAROLD G. WOLFF, JAMES D. HARDY, HELEN GOODELL. (Same Rm.).
Intracranial Aneurysms. BRONSON S. RAY. (Same Rm.).

9:00–12:00. *General Surgery.* Operative Clinic: General Surgery. FRANK GLENN and STAFF. (10th Fl. O.R.—Capacity 120).
Nonoperative Clinics: Follow-up Study of Treatment of Peptic Ulcer. WILLIAM A. COOPER. (College Auditorium, Rm. B-001—Capacity 350).
Bleeding Peptic Ulcer. CRANSTON HOLMAN. (Same Rm.).
Diagnosis of Stomach Lesions. WILLIAM A. COOPER. (Same Rm.).
Gastrojejunocolic Fistula. E. COOPER PERSON. (Same Rm.).
Carcinoma of the Head of the Pancreas. GARDNER CHILD. (Same Rm.).
Esophagogastrostomy; Follow-up of Cases. WILLIAM A. BARNES. (Same Rm.).

2:00–5:00. *Otolaryngology.* Operative Clinic: Surgery of the Paranasal Sinuses. SAMUEL F. KELLY. (10th Fl. O.R.—Capacity 30).

NEW YORK HOSPITAL
LYING-IN HOSPITAL

9:00–12:00. *Obstetrics and Gynecology.* Operative Clinics:
Tubal Plastic Operation for the Relief of Sterility. J. RANDOLPH GEPFERT. (Gyn. O.R., 2 Rms., Capacity 50).
Gynecological Operations. R. GORDON DOUGLAS. (Same Rm.).
Forceps Demonstration. JAMES A. HARRAR. (Same Rm.).

2:00–5:00. *Obstetrics and Gynecology.* Nonoperative Clinics:
Evaluation of Prophylactic Chemotherapy in Cesarean Section. R. GORDON DOUGLAS, ROBERT LANDESMAN. (Lecture Rm.—Capacity 100).

Labor and Delivery Following Cesarean Section. ARTHUR WILSON. (Same Rm.).
Pregnancy Following Salpingostomy. J. RANDOLPH GEPFERT. (Same Rm.).
Studies on Pain During Labor. CARL T. JAVERT, JAMES HARDY. (Same Rm.).
The Conservative Treatment of Endometriosis. ARTHUR V. GREELEY. (Same Rm.).
The Treatment of Massive Hemorrhage. JOHN T. COLE. (Same Rm.).
Vaginal Smears in Early Diagnosis of Carcinoma. GEORGE PAPANICOLAOU. (Same Rm.).

NEW YORK ORTHOPEDIC DISPENSARY AND HOSPITAL

8:30–12:00. *Orthopedic Surgery.* Operative Clinic. ALAN DE FOREST SMITH and STAFF. (O.R. 1 & 2—Capacity 12).
1:30–4:00. *Orthopedic Surgery.* General Orthopedic Clinic. HALFORD HALLOCK and STAFF. (7th Floor Assembly Room—Capacity 30).

NEW YORK POST-GRADUATE MEDICAL SCHOOL AND HOSPITAL

8:30–12:00. *Plastic Surgery.* Operative Clinic. GUSTAVE AUFRICHT. (O.R. 2—Capacity 10).
10:30–12:30. *General Surgery.* Nonoperative Clinic: Gall Bladder Disease, Common Duct Disord rs. R. FRANKLIN CARTER. (Erdmann Auditorium—Capacity 125).
2:00–5:00. *General Surgery.* Operative Clinic. H. E. CLARK. (O. R. 1—Capacity 10).
Operative Clinic. C. G. HEYD. (Surg. Amph.—Capacity 50).
2:00–5:00. *Gynecology.* Operative Clinic. M. N. HYAMS. (O.R. 5—Capacity 10).

PRESBYTERIAN HOSPITAL

8:00–12:00. *General Surgery.* Operative Clinics:
Surgery of the Breast. CUSHMAN D. HAAGENSON and STAFF. (Balcony Fl. 19, Main O.R. D-G—Capacity each 16).
Surgery of the Stomach. RUDOLPH N. SCHULLINGER, EDWARD L. HOWES. (Same Rm.).
Portacaval Anastomosis. ARTHUR H. BLAKEMORE (Same Fl. O.R. C.—Capacity 16).
2:00–4:00. *General Surgery.* Nonoperative Clinic: Certain Essential Features Suggested by 30 Year Follow-up in Carcinoma of the Breast. HUGH AUCHINCLOSS. (McCosh Amph., 18th Fl.—Capacity 142).
The Relation of Chronic Cystic Disease to Carcinoma of the Breast. ARTHUR PURDY STOUT. (Same Rm.).
The Problem of Early Diagnosis in Carcinoma of the Breast. CUSHMAN D. HAAGENSEN. (Same Rm.).
Roentgentherapy of Carcinoma of the Breast in Nonoperated Cases and as a Preoperative and Postoperative Treatment. MAURICE LENZ. (Same Rm.).
Surgery Versus Radiation in Carcinoma of the Breast. CUSHMAN D. HAAGENSEN. (Same Rm.).

PRESBYTERIAN HOSPITAL
BABIES HOSPITAL

9:00–11:00. *General Surgery.* Operative Clinic. MEMBERS OF THE STAFF. (O.R. 1 & 2, 9th Fl.—Capacity 5 each).
11:00–12:00. *General Surgery.* Nonoperative Clinic: The Surgical Treatment of Cyanotic Heart Disease. GEORGE H. HUMPHREYS. (Amph. 4th Fl.—Capacity 75).
2:00–4:00. *General Surgery.* Nonoperative Clinic:
Roentgen Findings in Intestinal Obstruction of the Newborn. JOHN CAFFEY. (Same Rm.).
Treatment of Acute Hematogenous Osteomyelitis. Comparison of Cases Treated with and without Penicillin. Follow-up. EDWARD B. SELF. (Same Rm.).
End-Results in Operations for Undescended Testicle. EDMUND N. GOODMAN. (Same Rm.).
Inguinal Hernia in Infancy and Childhood. Indications for Operation in Infancy. Follow-up. JOSE M. FERRER. (Same Rm.).

PRESBYTERIAN HOSPITAL
THE INSTITUTE OF OPHTHALMOLOGY

9:00–12:00. *Ophthalmology.* Nonoperative Clinic: External Diseases of the Eye. JOHN HUGHES DUNNINGTON and STAFF. (Lecture Rm. 7th Fl. Eye Inst.—Capacity 40).
2:00–4:00. *Ophthalmology.* Operative Clinic: Surgery of the Extraocular Muscles and Intraocular Operations. JOHN HUGHES DUNNINGTON. (O.R. Eye Inst.—Capacity 15).
Surgical Procedures in Glaucoma and Cataract. ALGERNON BEVERLY REESE and STAFF. (Same Rm.).

PRESBYTERIAN HOSPITAL
SECTION ON OTOLARYNGOLOGY

10:00–12:00. *Otolaryngology.* Nonoperative Clinic: Hearing Aid Fitting. MARY HINDMAN (Rm. 212, 1st Fl. Vanderbilt Clinic—Capacity 20).
Fenestration Follow-up. FRANZ ALTMANN. (Rm. 221, 1st Fl. Vanderbilt Clinic—Capacity 20).
Follow-up of Cases of Bilateral Abductor Paralysis of the Larynx. DEGRAAF WOODMAN. (Same Rm.).
1:30–5:00. *Otolaryngology.* Operative Clinic: Endoscopy. GEORGE BRIGHTON. (E.N.T. O.R. A and B—Capacity 16).
Fenestration. DEGRAAF WOODMAN. (Same Rm.).

PRESBYTERIAN HOSPITAL
SLOANE HOSPITAL FOR WOMEN

9:00–10:30. *Obstetrics and Gynecology.* Operative Clinic: Repair of Cystocele, Rectocele and Pelvic Floor. B. P. WATSON. (O.R. 17th Fl.—Capacity 20).
10:00–12:00. *Obstetrics and Gynecology.* Operative Clinic: Operation for Vesical Incontinence. JOHN H. BOYD. (Same Rm.).
2:00–3:00. *Obstetrics and Gynecology.* Nonoperative Clinic: Surgical Problems in Sterility. CHARLES L. BUXTON. (Lecture Rm. 17th Fl.—Capacity 40).
3:00–4:00. *Obstetrics and Gynecology.* Nonoperative Clinic: Interpretation of Endometrial Biopsy. E. T. ENGLE. (Same Rm.).
4:00–5:00. *Obstetrics and Gynecology.* Nonoperative Clinic: The Possible Carcinogenic Effect of Estrogens. S. GUSBERG. (Same Rm.).

PRESBYTERIAN HOSPITAL
SQUIER UROLOGICAL CLINIC

9:00–12:00. *Genitourinary Surgery.* Nonoperative Clinic:
Sulfonamide Mixtures in the Treatment of Genitourinary Disease. HARRY SENECA. (McCosh Amphitheater 18th Fl.—Capacity 142).
The Study of the Effect of Streptomycin on Renal Tuberculosis. Preliminary Report. JOHN K. LATTIMER. (Same Rm.).
Pathological Classifications of Adrenal Tumors. MEYER M. MELICOW. (Same Rm.).
Problems Encountered in the Diagnosis and Treatment of Congenital Anomalies of the Urinary Tract in Infants. JOHN N. ROBINSON, CHARLES T. HAZZARD. (Same Rm.).

2:00–4:00. *Genitourinary Surgery.* Operative Clinic: Surgical Operations. GEORGE F. CAHILL, GEORGE W. FISH and STAFF. (Balcony Fl. 19, Rms. C and D—Capacity 32).

Exhibit on Cystoscopic Equipment. (Rm. 1030, Babies Hosp., 10th Fl.—Capacity 32).

Exhibit of Urological Pathology Department. (Rm. 510, 15th Fl., Phys. & Surg. Bldg.—Capacity 32).

PRESBYTERIAN-NEW YORK ORTHOPEDIC HOSPITALS

Fracture Service

9:00–12:00. *Fractures and Traumatic Surgery.* Operative Clinic. FRACTURE SERVICE STAFF. (Main O.R. 19th Fl.—Capacity 8).

1:30–4:00. *Fractures and Traumatic Surgery.* Nonoperative Clinic: Shoulder Cuff Injuries and Recurrent Shoulder Dislocations with Follow-up Results. HARRISON L. MCLAUGHLIN. (Rm. 417, 16th Fl., Phys. & Surg. Bldg.—Capacity 100).

Results of Open and Closed Treatment of Potts' Fractures. BARBARA B. STIMSON. (Same Rm.).

ROOSEVELT HOSPITAL

9:00–12:00. *General Surgery.* Operative Clinic: Surgical Treatment of Lesions of the Colon: Subtotal Colectomy for Ulcerative Colitis. Rounds and Demonstration of Patients. HENRY W. CAVE and STAFF. (O.R. 1, 3, and 4—Capacity 42).

9:00–12:00. *Gynecology.* Operative Clinic: Gynecology. THOMAS G. PEIGHTAL, ARTHUR V. GREELY and STAFF. (O.R. 2—Capacity 14).

9:00–12:00. *General Surgery.* Operative Clinic: Cancer of the Breast: Radical Mastectomy. Rounds, Presentation of Cases and Illustration of Late Results. WILLIAM C. WHITE and STAFF. (O.R. 1–4—Total capacity 56).

11:00–12:00. *Gynecology.* Nonoperative Clinic: The Early Results of Malignancies of the Female Pelvic Organs: Prolapse of the Uterus, etc. (Conference Room 1—Capacity 50).

2:00–5:00. *General Surgery.* Nonoperative Clinic: Surgical Treatment of Lesions of the Colon: Sequelae, Complications and Late Results of the Surgical Treatment of Ulcerative Colitis. (Motion Pictures of Technique of Ileostomy and Colectomy. Presentation of Cases Showing Late Results). HENRY W. CAVE. (Same Rm.).

The Late Results of Carcinoma of the Thyroid Gland. JAMES E. THOMPSON. (Same Rm.).

Presentation of Cases Illustrating Interesting and Unusual Fractures (Illustrations). WILLIAM H. CASSEBAUM. (Same Rm.).

Nonunion in Fractures of the Femoral Neck, Case Presentations (Illustrations). M. BECKETT HOWORTH. (Conference Rm. 2—Capacity 50).

ST. LUKE'S HOSPITAL

8:30–1:00. *Genitourinary Surgery.* Operative Clinic. Urologic Survey. GEORGE HOCH and STAFF. (Lyle O.R.—Capacity 15).

11:00–1:00. *Orthopedic Surgery.* Clinical Demonstration: Pathological Demonstration of Internal Derangement of Knee Joint with End-Results. DAVID BOSWORTH, EDWARD WINANT. (Staff Conference Rm.—Capacity 60).

Trochanteric Fractures of Femur. Treatment and End-Results. FRED THOMPSON. (Same Rm.).

2:00–5:00. *Genitourinary Surgery.* Clinical Demonstrations: Follow-up on War Paraplegics. BORIS PETROFF.

Follow-up on Renal Tumors. JOHN TAYLOR. (Same Rm.).

Follow-up on Vesical Neck Obstructions to Abdominoperineal Resection. JAMES WAUGH. (Same Rm.).

Results of Transurethral Resection. GEORGE HOCH (presented by C. H. PLACE). (Same Rm.).

Combined Treatment of Transvesical Resection and Radiotherapy of Bladder Tumors. E. CRAIG COATES. (Same Rm.).

Renal Rickets. ALLEY. (Same Rm.).

Bilateral Hydronephrosis and Hydroureter. GEORGE HOCH. (Same Rm.).

ST. VINCENT'S HOSPITAL

9:00–12:00. *General Surgery.* Operative Clinic: General Surgery. RAYMOND P. SULLIVAN and ASSOCIATES. (O.R. A and B—Capacity 10 each).

9:00–12:00. *Neurosurgery.* Operative Clinic and Follow-up Study of the Treatment of Painful Phantom Limb by Removal of Sensory Cortex. C. G. DE GUITERREZ-MAHONEY. (O.R. C—Capacity 10).

10:00–12:00. *General Surgery.* Operative and Nonoperative Clinics: Surgery of Gastric and Duodenal Ulcer. GEORGE R. STUART, FRANK J. MCGOWAN, J. P. BRUCKNER. (O.R. D—Capacity 10).

2:00–5:00. *General Surgery.* Nonoperative Clinic: Round Table Discussion: Acute Pancreatitis. CONSTANTINE J. MAQUIRE. (Nurse's Auditorium—Capacity 150).

Reconstruction of Common Bile Duct. RAYMOND P. SULLIVAN, JOHN C. MURPHY. (Same Rm.).

Follow-up Report on 100 Cases of Fracture of Both Bones of the Legs. EDWARD V. DENNEEN, E. R. EASTON. (Same Rm.).

2:00–4:00. *Pathology.* Tissue Culture Demonstration of Malignancy. Display of Fresh Pathological Material. Display and Demonstration of System Diseases by Kodachrome Slides. A. ROTTINO and ASSOCIATES. (Nurse's Home Lab.—Capacity 30).

SLOANE HOSPITAL FOR WOMEN

(See Presbyterian Hospital)

U. S. MARINE HOSPITAL (STATEN ISLAND)

10:00–12:30. *General Surgery.* Nonoperative Clinics: Reconstruction Surgery of Face Deformities—Traumatic—Discussion and Case Presentation. Reconstruction Surgery of Hand—Discussion and Case Presentation. H. L. SKINNER and STAFF with DENTAL STAFF. (Doctor's Staff Rm.—Capacity 75).

10:00–12:30. *Obstetrics and Gynecology.* Operative and Nonoperative Clinic: Regional Block in Obstetrics and Gynecology. Film—Caudal Anesthesia. W. B. EDWARDS, C. L. HEBERT. (G.U. O.R.—Capacity 10).

12:30–1:30. Luncheon at the Hospital.

1:00–4:00. *Obstetrics and Gynecology.* Operative Clinics: Vaginal Plastic. Cesarean Section. Hysterectomy. EDWARD BULLARD, W. B. EDWARDS. (Surg. O.R. EF 4—Capacity 20).

2:00–4:00. *Ophthalmology.* Nonoperative Clinic: Acute Injuries of the Eye with Case Presentation. J. B. PEEBLES and STAFF. (Doctor's Staff Rm.—Capacity 75).

VETERANS ADMINISTRATION HOSPITAL

9:00–12:00. *General Surgery.* Operative Clinic. JOHN E. SULLIVAN, CARNES WEEKS. (Bldg. D, 2 Rms., 12th Fl.—Capacity 20).

9:00–12:00. *Thoracic Surgery.* Operative Clinic. RICHMOND MOORE, WALTER CRANDALL, AARON HIMMELSTEIN. (Bldg. D, 12th Fl., 1 Rm.—Capacity 10).

9:00–12:00. *Neurosurgery.* Operative Clinic. ERICH KRUEGER, FITZ CRAMER. (Bldg. D, 12th Fl., 1 Rm.—Capacity 10).

9:00–12:00. *Plastic Surgery.* Operative Clinic. CLARENCE R. STRAATSMA, ROBERT CLIFFORD. (Bldg. B, 5th Fl., 1 Rm.—Capacity 10).

9:00–12:00. *Otorhinolaryngology.* Operative Clinic. HENRY THALER. (Bldg. B, 5th Fl.—Capacity 5).

9:00–12:00. *Radiology.* Nonoperative Clinic: Radiotherapy: Lymphomas and Leucemia. BERNARD ROSWIT. (Bldg. C, Ground Fl.—Capacity 50).

9:30–11:30. *General Surgery.* Nonoperative Clinic: Medical Rehabilitation Clinic—Demonstrations. HARRY KESSLER, KARL HARPUDER, IRWIN D. STEIN, BERNARD STOLL, JACOB BYER. (Bldgs. B, C, & F—Capacity 20).

11:00–12:00. *General Surgery.* Nonoperative Clinic: Carcinoma of Pancreas. J. E. SULLIVAN. (Bldg. A, 3rd Fl.—Capacity 200).

1:30–3:30. *Thoracic Surgery.* Nonoperative Clinic: Conference. FRANK BERRY, RICHMOND MOORE, WALTER CRANDALL, AARON HIMMELSTEIN, and RESIDENT STAFF. (Same Rm.).

3:30–5:00. *General Surgery.* Nonoperative Clinic: Combined Medico-Surgical: Differential Diagnosis of Medical and Surgical Jaundice. FRANKLIN M. HANGER, ALLEN O. WHIPPLE, FREDERIC W. BANCROFT, BERNARD STRAUS, CHARLES A. FLOOD, JOSEPH POST. (Same Rm.).

EXHIBITS
(Bldg. A, 3rd Fl.)

Anesthesia Section
Bronchogenic Carcinoma: Review of 600 Cases
Intracranial Neoplasms
Residency Training Program
Organization of Surgical Division
Results of Vagus Resection
Pathology Section
Medical Illustrations Laboratory
Plastic Surgery
Prosthetic Appliances

CLINICS IN BROOKLYN—LONG ISLAND HOSPITALS

Wednesday

BROOKLYN EYE AND EAR HOSPITAL

9:00–12:00. *Ophthalmology and Otolaryngology.* Nonoperative Clinic: Late Results in Detachment of Retina. Late Results in Cataract Extraction. Late Results in Fenestration. Late Results in Dacryocystorhinostomy. Late Results in Ptosis. Motion Picture Demonstration in Color on Basic Anatomy of the Visual Pathway as Shown by Animation and Dissection. JACOB GOLDSTEIN, N. P. TANTILLO, JOHN P. BAKER, B. WELT, A. B. RIZZUTI, C. R. NOBACK, Ph.D. (Solarium—Capacity 40).

2:00–4:00. *Ophthalmology.* Operative Clinic. E. C. PLACE and STAFF. (7th Fl. O.R. 1–4—Capacity 20).

CUMBERLAND HOSPITAL

9:00–12:00. *General Surgery.* Operative Clinic: Male Surgery. JOHN A. TIMM and STAFF. (O.R. 1, 2 & 3—Capacity 20).

9:00–12:00. *General Surgery.* Operative Clinic: Gastrectomy for Cancer of the Stomach with Discussion of Late Results. JOHN J. GAINEY and STAFF. (Auditorium—Capacity 300).

Nonoperative Clinic: Ward Rounds with Discussion of Surgical Gall Bladder with Late Results. JOHN A. TIMM and STAFF. (Surg. Wards—Capacity 15).

Nonoperative Clinic: Ward Rounds with Discussion of Surgery in Large Bowel Malignancy. HOWARD T. BLAIR. (Same Rm.).

12:30–1:30. Luncheon at the Hospital.

2:00–5:00. *General Surgery.* Nonoperative Clinics: Arteriovenous Fistula with Follow-up. JOSEPH I. ANTON. (Auditorium—Capacity 300).

Common Duct Atresia. JOHN A. TIMM. (Same Rm.).

Congenital Esophageal Atresia. SAUL F. LIVINGSTON. (Same Rm.).

Traumatic Evisceration with Follow-up. MARTIN LUTZ. (Same Rm.).

Lymphogranuloma Inguinale (Venereum) of Uterus. Follow-up. SILIK H. POLAYES.

Thoracic Surgery in General Hospital. WILLIAM H. FIELD.

Common Duct Atresia. JOHN A. TIMM.

Congenital Esophageal Atresia. SAUL F. LIVINGSTON.

Traumatic Evisceration with Follow-up. MARTIN LUTZ.

JEWISH HOSPITAL

8:00–10:00. *General Surgery.* Operative Clinics. HENRY LOURIA and STAFF. (Amph. 1—Capacity 25).

Operative Clinic. RUDOLPH NISSEN and STAFF. (Amph. 2—Capacity 25).

10:00–12:00. *General Surgery.* Nonoperative Clinic: Curare and Endotracheal Anesthesia in Poor Risk Patients. IRVING M. PALLIN. (Auditorium—Capacity 200).

Treatment of Postoperative Phlebothrombosis with Subcutaneous Heparin. LEO LOEWE, EDWARD HIRSCH. (Same Rm.).

"Bleeding Nipple." SAUL F. LIVINGSTON. (Same Rm.).

Acute Mediastinitis. LEW A. HOCHBERG. (Same Rm.).

Endorrhaphy as Surgical Procedure in the Treatment of Giant Air Cysts of the Lung. RUDOLPH NISSEN. (Same Rm.).

External Ophthalmoplegia Associated with Hyperthyroidism. VICTOR WORONOV. (Same Rm.).

The Diagnosis and Surgical Treatment of Thyroiditis. ARTHUR A. WEINBERG. (Same Rm.).

Malignant Tumors of the Thyroid Gland Diagnosis, Management and End-Results. FRANK TELLER. (Same Rm.).

Surgical Management of Goiter. HENRY W. LOURIA. (Same Rm.).

Operation for Carcinoma of the Colon Involving as well Adjacent Viscera. GEORGE STRENGER. (Same Rm.).

2:00–5:00. *General Surgery.* Nonoperative Clinic: Two Unusual Cases of Sciatic Pain. S. A. BERNSTEIN. (Same Rm.).

Early Dysplasia of the Hip. Diagnosis and Treatment. M. T. KOVEN. (Same Rm.).

The Conservative Management of Slipping of the Upper Femoral Epiphysis. L. S. NELSON. (Same Rm.).

The Use of the Austin Moore Plate and Its Modifications in High Osteotomy of the Femur. B. KOVEN. (Same Rm.).

Reconstruction Key Gliding Mechanism of Upper Extremity. JOSEPH E. MILGRAM. (Same Rm.).

Composition of Bone in Relation to Blood and Diet. A. E. SOBEL. (Same Rm.).

The Application of Laminography to Some Orthopedic Problems. B. S. EPSTEIN. (Same Rm.).

KINGS COUNTY HOSPITAL

9:00–11:00. *Plastic Surgery.* Operative Clinics: Cleft Palate. Skin Graft of Burns. WALTER COAKLEY and STAFF. (B Bldg. O.R. 1—Capacity 15).

9:00–11:00. *Plastic Surgery.* Nonoperative Clinic: Presentation of Cases, Ward Walk.

Acute Burns (Lye, Acid, Hot Water, Electricity and Fire).
Hump and Saddle Noses.
Cleft Lip and Palate.
Contracted Scars.
Web Fingers.
Hemangioma and Lymphangioma of Lip.
Jug-handle Ears.
Depressed Fractures—(a) of Malar Bone; (b) of Zygomatic Processes; (c) of Infraorbita Ridge; (d) Fractures of Mandible with and without Loss of Substance; (e) Fractures of Superior Maxilla.
Skin Grafts—(a) Split-skin, (b) Tube Grafts.
Exhibition of Moulages, Photographs and X-rays of Facial Bone Fractures.
Animated Motion Pictures: Reconstructive Surgery of the Bones of the Face. Cleft Palate Operation. (O.P.D. Bldg., Lecture Rm.—Capacity 100).

2:00–5:00. *Plastic Surgery.* Nonoperative Clinic: Presentations. WALTER COAKLEY. (Same Rm.).
Experiences in the Treatment of 1500 Severe Burns. WALTER COAKLEY. (Same Rm.).
Results of Skin-Grafting in Chronic Leg Ulcers. DAVID TEPLITSKY. (Same Rm.).
Skin-Grafting of Burns. JOSEPH CALTAGIRONE. (Same Rm.).
Rhinoplastic Procedures. (Same Rm.).

LONG ISLAND COLLEGE HOSPITAL

9:00–1:00. *General Surgery.* Operative Clinics: Thyroidectomy and Other Procedures. EMIL GOETSCH and STAFF. (Surg. Amphitheater—Capacity 100).
Nonoperative Clinics: (Same Rm.).
Chronic Thyroiditis. EMIL GOETSCH. (Same Rm.).
Rôle of Thiouracil and Its Derivatives in the Preparation of the Critically Ill Hyperthyroid Patient. ARTHUR GOETSCH.
The Pressure Case as a Problem in Thyroid Surgery. ALBERT J. RITZMANN.
Surgical Treatment of Colon Carcinoma. BENJAMIN CISSEL.
Radical Mastectomy for Breast Malignancy; Rodman's Method. GAETANO DEYOANNA.
Spontaneous Rupture of Bile Ducts Following Cholecystectomy. HAROLD LA ROE.
Postoperative Management of Thoracic Surgery Patients. E. K. JOHNSON.
Exteriorization of Ileocecal Region for Obstructing Lesions in Infants and Children. ROBERT MAINZER.
(The Departments of Ophthalmology and Otolaryngology will conduct their programs at the Brooklyn Eye and Ear Hospital.)

METHODIST HOSPITAL

8:00–12:00. *General Surgery.* Operative Clinic. PIERRE A. RENAUD, JOHN A. TIMM and STAFF. (Main O.R. 1–6—Capacity 50).

Genitourinary Surgery. Operative Clinic. HOWARD T. LANGWORTHY and STAFF. (5th Fl. Urology O.R.—Capacity 10).

12:00–1:00. Luncheon at Hospital.

2:00–5:00. *General Surgery.* Nonoperative Clinic: Symposium on General Surgery and Specialties. ARTHUR H. BOGART, Moderator.
Surgery of the Gastrointestinal Tract in the Newborn. C. D. SAWYER. (Nurse's Auditorium—Capacity 300).
The Meynen Operation for Inguinal Hernia. E. MARTINSON. (Same Rm.).
Hyperpyrexia in Surgery. A. VOSSELER. (Same Rm.).
Management of Acute Cholecystitis. H. F. GRAHAM. (Same Rm.).
Acute Pancreatitis—A Ten Year Survey. M. CARMER. (Same Rm.).
Supracondylar Fractures of Humerus. H. P. LANGE. (Same Rm.).
Follow-up on Unusual Genitourinary Cases. H. T. LANGWORTHY. (Same Rm.).
Tuberculosis in Polycystic Kidney. R. MULE. (Same Rm.).
Care of Genitourinary Tract in Paraplegics. O. P. SCHOENEMANN. (Same Rm.).
Thoracoabdominal Injuries. E. MARTINSON. (Same Rm.).
Demonstration of Pathology. J. HOWE. (Same Rm.).
Demonstration of roentgen-rays. G. W. CRAMP. (Same Rm.).

ST. JOHN'S HOSPITAL

8:00–10:00. *General Surgery.* Operative Clinic: Thyroidectomy for Toxic Diffuse Goiter. MERRIEL N. FOOTE and STAFF. (O.R. A, B & C—Total Capacity 90).
Patient Prepared with Propylthiouracil. Subtotal Gastric Resection for Duodenal Ulcer. GEORGE MARSH AND STAFF. (Same Rm.).

8:00–10:00. *Gynecology.* Operative Clinic: Manchester-Fothergill Operation. Vaginal Hysterectomy. CHARLES MUELLER, W. LAPP and STAFF. (Same Rm.).

10:00–12:00. *General Surgery.* Nonoperative Clinic:
Demonstration of Unusual X-ray Findings and Problems in Diagnosis—Informal Discussion. W. TERMON (X-ray Dept. 5th Fl. E.—Capacity 15).
Demonstration of Unusual Gross Pathological Specimens—Informal Discussion. R. GETTINGER (Wing Tissue Lab. 5th Fl.—Capacity 15).
Ward Rounds—Gynecology. C. MUELLER (East Wing Women's Surg. 1st Fl.—Capacity 20).
Ward Rounds—General Surgery Including Thoracic Surgery, Surgery of the Hand, Gastrointestinal Surgery, Orthopedic Surgery, Urology. M. FOOTE, E. K. JOHNSON, G. REITZ, G. MARSH, R. WARREN, A. MICHELE, A. HARRIS (Male Surg., 2nd Fl., West Wing and Female Surg., 1st Fl.—Capacity 40).

1:30–4:30. Round Table Discussion:
The Present Status of Propylthiouracil in Toxic Goiter. MERRILL N. FOOTE. (Nurse's Lecture Hall—Capacity 150).
Open Surgical Resection for Carcinoma of the Dome of the Bladder as a Treatment of Choice—End-Results. A. HARRIS. (Same Rm.).
The Clinical Application of Cardiopulmonary Physiology in Thoracic Surgery. ELGIE JOHNSON. (Same Rm.).
Evaluation of Therapeutic and Diagnostic Paravertebral Block in Vascular Diseases of the Extremities. LEONID WATTER. (Same Rm.).
Surgical Lesions of the Hand, Follow-up Results. GEORGE B. REITZ. (Same Rm.).
(a) Postpartum Eclampsia Associated with Hydatidiform Mole—End-Results; (b) Geriatric Gynecological Surgery—End-Results. CHARLES MUELLER. (Same Rm.).
Shoulder Pain—Diagnosis and discussion of Operative Treatment. ROBERT WARREN, ARTHUR MICHELE. (Same Rm.).
The Value of Detergent (Areusol O.T.) in Antibiotic Therapy. E. J. GRACE. (Same Rm.).

CLINICS IN NEW YORK HOSPITALS

Thursday

BABIES HOSPITAL
(See Presbyterian Hospital)

BELLEVUE HOSPITAL—FIRST (COLUMBIA UNIVERSITY) SURGICAL DIVISION

8:00–12:00. *Thoracic Surgery.* Operative Clinic. FRANK B. BERRY and STAFF. (C & D Bldg. 5th Fl.—Capacity 10).

2:00–4:30. *Thoracic Surgery.* Nonoperative Clinic. FRANK B. BERRY and STAFF. (C & D Bldg. Stewart Amph.—Capacity 100).

Suppurative Diseases of the Lung. WALTER B. CRANDELL.

Cardiopulmonary Physiology. RICHARD RILEY, AARON HIMMELSTEIN.

Surgery of Tuberculosis. JOHN MAXWELL CHAMBERLAIN.

Surgical Treatment of Chronic Spontaneous Pneumothorax. HERBERT C. MAIER.

Carcinoma of the Lung. ADRIAN LAMBERT.

BELLEVUE HOSPITAL—THIRD (NEW YORK UNIVERSITY) SURGICAL DIVISION

8:00–10:00. *Neurosurgery.* Operative Clinic. Thoracolumbar Sympathectomy for Hypertension. H. M. WERTHEIM, JOHN H. MULHOLLAND and STAFF. (Surg. O.R. K-5—Capacity 5).

8:00–10:00. *General Surgery.* Operative Clinics (Operating Rm. K-5 & K-6; Psychiatric Surgical Operating Room). JOHN H. MULHOLLAND and STAFF.

Thyroidectomy. JOHN A. LAWLER (O.R. A).

Anterior Resection of the Rectum for Carcinoma. SAMUEL STANDARD (O.R. B).

Section of Sphincter of Oddi for Pancreatitis. JOHN MULHOLLAND, HENRY DOUBILET (O.R. C).

Lumbar Sympathectomy for Peripheral Vascular Disease. WALLACE B. MURPHY (Operating Room D).

Murray-Brackett Osteotomy for Unlimited Fracture of Neck of Femur. IRWIN S. SIRIS (Bone Rm. K-6—Capacity 5).

10:30–1:00. *General Surgery.* Nonoperative Clinics (I & K Bldg., Stewart Amph.).

Results of Section of Sphincter of Oddi for Pancreatitis. HENRY DOUBILET.

Rôle of Sulfadiazine in Thyrotoxic Patients. JOHN A. LAWLER.

Results and Follow-up in Carcinoma of the Rectum Treated with Anterior Resection. SAMUEL STANDARD.

Pulmonary Complications in Surgery. WALTER CRANDELL.

Fluid Compartments in Depleted Surgical Patients and Changes During Repletion. Co TUI.

General Principles of Reconstructive Plastic Surgery of the Face. JOHN M. CONVERSE.

Program of Postgraduate Training in Surgery at New York University College of Medicine. JOHN H. MULHOLLAND.

8:30–12:00. *Gynecology.* Operative Clinic. WILLIAM E. STUDDIFORD and STAFF (K-6 O.R.—Capacity 5).

Total Hysterectomy for Fibroid. CLAUDE E. HEATON.

Unusual Cases of Dysmenorrhea in Young Women—A Report of 2 Cases (paper).

Fascial Transplant for Relief of Stress Incontinence in Patients with Previous Operations. WILLIAM E. STUDDIFORD.

Pulmonary Complications in Surgery. WALTER CRANDELL.

Fluid Compartments in Depleted Surgical Patients and Changes During Repletion. Co TUI.

General Principles of Reconstructive Plastic Surgery of the Face. JOHN M. CONVERSE.

Program of Postgraduate Training in Surgery at New York University College of Medicine. JOHN M. MULHOLLAND.

2:00–5:00. *Otorhinolaryngology.* Operative Clinic (K-6 O.R.) Laryngeal Surgery. JOHN F. DALY and STAFF.

2:00–5:00. *Anesthesia.* Demonstrations (K-6 O.R.). Therapeutic Nerve Block Clinic. Anesthesia and Shock. E. A. ROVENSTINE and STAFF.

2:00–5:00. Rehabilitation and Physical Medicine for Paraplegiae. Demonstration of Programs for the Rehabilitation of Paraplegic Patients. GEORGE DEAVER. (Wards F, G-3, F & G Bldg.—Capacity 25).

2:00–4:30. Case Demonstrations. IRWIN E. SIRIS, THOMAS J. GALVIN, EDGAR H. BATES, JESSIE W. MAHONEY, WILLIAM MACKLER. (I & K Bldg. Stewart Amph.—Capacity 100).

BETH ISRAEL HOSPITAL

9:00–12:00. *Gynecology.* Operative and Nonoperative Clinic. HENRY FALK and STAFF. (Main O.R., 12th Fl.—Capacity 6; 3rd Fl. Lecture Rm.—Capacity 30).

2:00–5:00. *Anesthesia.* Nonoperative Clinic: Clinical Demonstrations—Film on Hemorrhage and Shock. S. G. HERSHEY and STAFF. (3rd Fl. Lecture Rm.—Capacity 30).

FLOWER AND FIFTH AVENUE HOSPITALS

9:00–11:00. *Neurosurgery.* Operative Clinic: Lumbar Sympathectomy—Technique of Block. S. THOMAS GLASSER, DONALD E. BRACE. (Main O.R., 8th Fl.—Capacity 12).

11:00–12:00. *General Surgery.* Nonoperative Clinic: Hand Infections. JAMES M. WINFIELD, JOHN HERRLIN. (Auditorium—Capacity 300).

Nonoperative Clinic: Diseases of Vascular System. FREDERIC W. BANCROFT. (Same Rm.).

2:00–3:00. *General Surgery.* Nonoperative Clinic: Exhibit of Postgraduate School—Regional Ileitis. JOSEPH H. FOBES. (Same Rm.).

3:00–5:00. *General Surgery.* Nonoperative Clinic: Functional Concept of the Internal Ring—Surgical Application in the Repair of Inguinal Hernia—Operating Room or Cadaver. J. CLIFFORD HAYNER. (7th Fl. College Anatomy Lab.—Capacity 100).

HARLEM HOSPITAL

9:00–12:00. *Traumatic Surgery.* Nonoperative Clinic: Trauma Symposium. Ward Rounds. L. T. WRIGHT (Wards 2A, 2B, Main Bldg. and Conference Rm., 5th Fl. Women's Pavilion).

Head and Neck Injuries. D. H. SMITH, J. GREENE, S. SHENKMAN. (Same Rm.).

Fractures of the Extremities with Late Results. A. C. LOGAN, W. A. FREEMAN, A. PRIVITERA, C. DI LORENZO. (Same Rm.).

2:00–5:00. *Obstetrics.* Nonoperative Clinic: Obstetrical Symposium. Obstetrical Emergencies; Toxemia of Pregnancy. F. A. KASSEBOHM, A. C. POSNER, J. KURZROCK, M. SCHREIBER, N. PLESCHETTE. (Conference Rm. 5th Fl., Women's Pavilion—Capacity 100).

HOSPITAL FOR JOINT DISEASES

9:00–12:00. *Orthopedic Surgery.* Operative Clinic. HARRY SONNENSCHEIN and STAFF.
Transcervical Osteotomy for Slipped Femoral Epiphysis. A. KENIN. (Surg. Amph.—Capacity 50).
Femoral Shortening for Inequality of Leg Length. B. J. MINTZ. (Same Rm.).
Nonoperative Clinic: Transcervical Osteotomy. A. KENIN.
Equalization of Leg Length. B. J. MINTZ.
Tendon Transplantation for Radial Nerve Paralysis. H. ALTMAN. (Lecture Rm.—Capacity 60).
2:00–3:00. *Orthopedic Surgery.* Nonoperative Clinic: Demonstration: X-Rays of Primary Bone Tumors. MAURICE POMERANZ. (Lecture Rm.—Capacity 50).
2:00–4:00. *Ophthalmology.* Operative Clinic: Operations on the Eye. JAMES W. SMITH. (Small O.R.—Capacity 5).
2:00–4:00. *General Surgery.* Operative Clinic: Surgery of the Hand. LEO MAYER. (Surg. Amph.—Capacity 50).
3:00–4:00. *Orthopedic Surgery.* Nonoperative Clinic: Surgical Anatomy: Mechanism of Motion of Thumb. EMANUEL KAPLAN. (Lecture Rm.—Capacity 50).

HOSPITAL FOR SPECIAL SURGERY

8:30–12:00. *General Surgery.* Operative Clinic. Operations by General Surgical Department. F. BEEKMAN and STAFF. (Main O.R.—Capacity 32).
9:00–12:00. *Orthopedic Surgery.* Adult Orthopedic Problems. PHILIP D. WILSON, R. STEPHENS and STAFF. (Lecture Rm.—Capacity 100).
2:00–5:00. *Orthopedic Surgery.* Nonoperative Clinic: Hip Problems.
Treatment of Fractures of Femoral Neck in the Aged. FREDE-RICK VON SAAL. (Same Rm.).
Anterior Wedge Osteotomy for Slipped Femoral Epiphyses. LEWIS CLARK WAGNER, (Same Rm.)
Trochanteric Arthroplasty. PHILIP D. WILSON. (Same Rm.).
Transplantation of External Oblique for Paralysis of Hip Abductors. LOWELL THOMAS, T. CAMPBELL THOMPSON, L. RAMSAY STRAUB. (Same Rm.).
Treatment of Congenital Dislocation of the Hip with Anteversion. FRANCIS CARR. (Same Rm.).
Idiopathic Aseptic Necrosis of the Hip in Adults. WILLIAM COOPER. (Same Rm.).

LENOX HILL HOSPITAL

9:00–12:00. *General Surgery.* Nonoperative Clinic: Rehabilitation and Physical Medicine. GEORGE G. DEAVER. (Einhorn Auditorium—Capacity 450).
2:00–5:00. *General Surgery.* Nonoperative Clinics:
The Anti Coagulants and Their Use in Surgical Practice. CARL REICH. (Einhorn Auditorium—Capacity 450).
RH Factor and Blood Bank Demonstration. JACOB GEIGER. (Same Rm.).

LYING-IN HOSPITAL

(See New York Hospital)

MANHATTAN EYE, EAR AND THROAT HOSPITAL

9:00–12:00. *Ophthalmology.* Operative Clinic: Cataract Extraction; Corneal Transplant; Dacryocystorhinostomy. G. BONACCOLTO. (Eye O.R., 6th Fl.—Capacity 5).
10:00–12:00. *Ophthalmology.* Nonoperative Clinic: Laboratory Demonstration: Bacteriology of the Eye. F. C. KEIL, JR. (Path. Lab., 6th Fl.—Capacity 5).
10:00–12:00. *Otolaryngology.* Nonoperative Clinic: Demonstration: Results in Cases of Arytenoidectomy. J. D. KELLY. (Kelly Clinic, 2nd Fl.—Capacity 24).
10:00–12:00. *Ophthalmology and Otorhinolaryngology.* Nonoperative Clinic: Plastic Surgery Clinic: Conference on End-Results; Lantern Slides and Motion Pictures. J. M. CONVERSE, BYRON SMITH. (Conference Rm. 2nd Fl.—Capacity 70).
1:00–4:00. *Ophthalmology and Otorhinolaryngology.* Operative Clinic: Plastic Surgery. BYRON SMITH, J. M. CONVERSE. (O.R., 6th Fl.—Capacity 5).
2:00–4:00. *Ophthalmology.* Operative Clinic: Corneal Graft R. T. PATON. (Same Rm.).
2:00–4:00. *Ophthalmology.* Nonoperative Clinic: Demonstration: Vitreous Substitution; Technique and End-Results. MILO FRITZ. (Eye Bank Lab., 10th Fl.—Capacity 12).
2:00–4:00. *Ophthalmology and Otolaryngology.* Nonoperative Clinic: Radiology Clinic: Radiation Therapy in Ophthalmology. Results of Radiation in Laryngeal Tumors. MAURICE LENZ, J. R. FREID, C. OKRAINETZ. (Radiotherapy Dept., 3rd Fl.—Capacity 12).
2:00–4:00. *Otorhinolaryngology.* Nonoperative Clinic: Demonstration: Radiation of Hypertrophied Lymphoid Tissue of Nasopharynx. H. D. TATERKA. (Kelly Clinic, 2nd Fl.—Capacity 24).
2:00–5:00. *Otolaryngology.* Operative Clinic: Arytenoidectomy Operation. J. D. KELLY. (O.R. 6th Fl.).
3:00–5:00. *Ophthalmology.* Nonoperative Clinic: Demonstration: Postoperative Corneal Graft Cases. R. T. PATON. (Conference Rm. 2nd Fl.—Capacity 70).

MEMORIAL HOSPITAL

9:00–12:00. *Tumor Surgery.* Nonoperative Clinic: Discussion: Five Year Survivals in Lymphomas and Leucemias. LLOYD F. CRAVER. (Auditorium—Capacity 250).
The Use of Newer Nitrogen Mustard Compounds in the Treatment of Lymphomas and Leucemias. JOSEPH BURCHENAL. (Same Rm.).
Nitrogen Mustard As a Palliative Agent in Cancer. DAVID KARNOFSKY. (Same Rm.).
The Rôle of Radioactive Iodine in Thyroid Cancer. JACK TRUNELL. (Same Rm.).
Reasons for Delay in the Diagnosis of Cancer. JOHN E. LEACH, GUY ROBBINS. (Same Rm.).
Preoperative and Postoperative Care for Cancer of the Lung. JOHN E. LEACH, HENRY DIAMOND. (Same Rm.).
Study of Diagnostic Methods and Treatment of Cancer of the Lung. JOHN S. LADUE. (Same Rm.).
The Internist's Rôle in Cancer. WILLIAM H. LEWIS, JR. (Same Rm.).
Treatment of Shock. HENRY TAGNON. (Same Rm.).
2:00–3:30. *Tumor Surgery.* Nonoperative Clinic: Follow-up Results in Different Types of Breast Cancer. FRANK ADAIR. (Same Rm.).
3:30–5:00. *Tumor Surgery.* Follow-up Results in Patients with Metastatic Carcinoma of the Breast with Endocrine Therapy. FRANK ADAIR. (Same Rm.).

METROPOLITAN HOSPITAL

9:00–12:00. *General Surgery.* Nonoperative Clinic: Preoperative and Postoperative Treatment. JAMES M. WINFIELD and STAFF, and LOUIS R. KAUFMAN, JOHN HERRLIN, EDWARD J. MCCABE. (Conference Rm.—Capacity 60).
General Discussion.
Fluid Balance. JOHN HERRLIN. (Same Rm.).

Early Ambulation and Early Exercise. EDWARD J. MCCABE. (Same Rm.).

2:00–4:00. *Obstetrics and Gynecology.* Nonoperative Clinics: Interesting Ovarian Tumors. W. B. SILBERBLATT. (Same Rm.).

Ectopic Pregnancy with Especial Reference to Posterior Colkotomy. JOHN E. TRITSCH. (Same Rm.).

2:00–4:00. *Orthopedic Surgery and Fractures.* Nonoperative Clinic: Demonstration of End-Results of Injuries to Lower Extremities. MILTON J. WILSON and STAFF. (Conference Rm., Welfare Dispensary, 80th E. and East End Ave.—Capacity 50).

MONTEFIORE HOSPITAL

2:00–5:00. *Thoracic Surgery.* Operative and Nonoperative Clinic. ARTHUR H. AUFSES, ISIDOR KROSS. (O.R.—Capacity 10).

MOUNT SINAI HOSPITAL

8:30–12:00. *General Surgery.* Operative Clinic: Subtotal Gastrectomy and Infradiaphragmatic Vagotomy for Duodenal Ulcer with Stenosis. RALPH COLP. (Surg. Amph.—Capacity 100).

Nonoperative Clinics: Presentations: Physiologic Differences between Gastric and Duodenal Ulcers. FRANK HOLLANDER. (Same Rm.).

A Rational Approach to the Surgery of Juxta-Esophageal Ulcer. LEONARD DRUCKERMAN. (Same Rm.).

Problems in the Treatment of Gastrojejunocolic Fistula. PERCY KLINGENSTEIN. (Same Rm.).

Follow-up Results in Subtotal Gastrectomy for Gastric and Duodenal Ulcer. SIGMUND MAGE. (Same Rm.).

Indications for Vagotomy in Peptic Ulcer. RALPH COLP. (Same Rm.).

2:00–5:00. *Otorhinolaryngology.* Operative and Nonoperative Clinic: Inflammatory and Neoplastic Diseases of the Frontal Sinuses. RUDOLPH KRAMER, MORRIS BENDER, IRVING GOLDMAN, JOSEPH DRUSS, SAMUEL ROSEN and STAFF. (Same Rm.).

Carcinoma of the Larynx and Results, Treatment by Radiation Therapy. RUDOLPH KRAMER, MORRIS BENDER, IRVING GOLDMAN, JOSEPH DRUSS, SAMUEL ROSEN and STAFF. (Same Rm.).

NEW YORK EYE AND EAR INFIRMARY

10:00–12:00. *Otolaryngology and Ophthalmology.* Fundamentals of X-ray Diagnosis in Ophthalmology and Otolaryngology. IRVING SCHWARTZ. (X-ray Viewing Rm.—Capacity 10).

2:00–3:00. *Ophthalmology and Otorhinolaryngology.* Discussion: Allergy in Eye, Ear, Nose and Throat. HOWARD WESTCOTT. (Allergy Clinic—Capacity 20).

2:00–4:00. *Otorhinolaryngology.* Demonstration: Equipment Including the Biplanor Fluoroscope in the Bronchoscopic Clinic. MAXWELL D. RYAN. (Bronchoscopic Clinic—Capacity 20).

3:30–4:30. *Otorhinolaryngology.* Demonstration: Methods —Allergy. J. H. BARNARD. (Allergy Clinic—Capacity 20).

2:00–5:00. *Ophthalmology.* Demonstration: Postoperative Treatment. R. MEEK and W. JOHNSON and ASSOCIATES. (Clinic Rms.—Capacity 30).

Diagnostic Studies with Special Reference to Muscle Studies. T. BOYES and ASSOCIATES. (Same Rm.)

Surgery with Special Reference to Lacrimal Apparatus. B. PAYNE and R. MCCLANAHAN and ASSOCIATES. (Eye O. R.—Capacity 10).

Routine clinics of the New York Eye and Ear Infirmary will be held as usual during this period; all Fellows of the American College of Surgeons are cordially invited to attend.

NEW YORK HOSPITAL

9:00–12:00. *Plastic and Faciomaxillary Surgery.* Operative and Nonoperative Clinic: One Stage Push Back Operation for Congenitally Short Palate. Miscellaneous Case Presentations. HERBERT CONWAY. (Room F639—Capacity 70).

Speech Therapy in Cases of Cleft Palate. E. FREUD. (Same Rm.).

End-Results Following Radical Breast Amputation With Split Skin Grafting. CHARLES NEWMAN. (Same Rm.).

Demonstration of Technique of Tattooing Skin Grafts and Port Wine Stains. HERBERT CONWAY, FRANK ELISOU. (Same Rm.).

9:00–12:00. *Ophthalmology.* Operative Clinic: Intracapsular Cataract; Glaucoma; Strabismus. JOHN MCLEAN and STAFF. (10th Fl. O.R. 1 Rm.—Capacity 30).

2:00–5:00. *General Surgery.* Operative and Nonoperative Clinic. FRANK GLENN and STAFF.

Surgical Treatment of Acute Cholecystitis. FRANK GLENN. (College Auditorium, Rm. B001—Capacity 350).

Pathology of Acute Cholecystitis. N. C. FOOTE.

Complications of Acute Cholecystitis. S. W. MOORE.

Diverticulitis of Large Bowel, Follow-up Studies. GARDNER CHILD.

Follow-up Studies of Polyps of the Large Intestine. S. W. MOORE.

Demonstration and Exhibit. Surgical Pathological Laboratory. N. C. FOOTE.

2:00–5:00. *General Surgery.* Demonstration: Surgical Anatomy. Anatomy Involved in Lumbar and Thoracolumbar Sympathectomy. ERNEST LAMPE. (Rm. A-132—Capacity 15).

2:00–5:00. *Otorhinolaryngology.* Nonoperative Clinic: Case Presentations and Postoperative Result. Laryngofissure, Carcinoma of Antrum. Mastoiditis with Petrositis and Meningitis. Acute Frontal Sinusitis with Meningitis. ARTHUR PALMER and ASSOCIATES. (Rm. F639—Capacity 70).

NEW YORK HOSPITAL LYING-IN HOSPITAL

9:00–12:00. *Gynecology.* Operative Clinic: Gynecological Operations. H. J. STANDER. (Gyn. O. R., 2 Rms.—Capacity 50).

2:00–5:00. *Obstetrics and Gynecology.* Nonoperative Clinics: Late Results in Patients with Recurrent Abortion. CARL T. JAVERT. (Lecture Rm.—Capacity 100).

Eclampsia. H. J. STANDER. (Same Rm.).

Treatment of Endocervicitis. RALPH W. GAUSE. (Same Rm.).

X-Ray Pelvimetry. CHARLES M. MCLANE. (Same Rm.).

Missed Abortion. FRANK SMITH. (Same Rm.).

NEW YORK ORTHOPEDIC DISPENSARY AND HOSPITAL

9:00–12:00. *Orthopedic Surgery.* Rounds. End-Result Conference. ALAN DE FOREST SMITH. (7th Fl. Assembly Rm.—Capacity 30).

Osteoarthritis of the Hip. FRANK STINCHFIELD. (Same Rm.).

Slipping of the Upper Femoral Epiphysis. M. B. HOWARTH. (Same Rm.).

1:30–4:00. *Orthopedic Surgery.* General Orthopedic Clinic. LEONIDAS A. LANTZOUNIS and STAFF. (Same Rm.).

Scoliosis Clinic. WILLIAM H. VON LACKUM. (Same Rm.).

NEW YORK POST-GRADUATE MEDICAL SCHOOL AND HOSPITAL

8:30–9:30. *Neurosurgery.* Operative Clinic: Diagnostic Procedures. J. A. MACLEAN. (O.R. 4—Capacity 10).

8:30–11:30. *Urology.* Operative Clinic. CLARENCE BANDLER. (O.R. 5—Capacity 10).

8:30–12:00. *Tumor Surgery.* Operative Clinic. H. W. MEYER. (O.R. 2—Capacity 10).

8:30–12:00. *General Surgery.* Operative Clinic. E. V. DENEEN. (O.R. 1—Capacity 10).

Operative Clinic. R. FRANKLIN CARTER. (Surg. Amph. —Capacity 50).

10:30–12:30. *General Surgery.* Nonoperative Clinic: Case Presentations of Operative Procedures in Gastroduodenal Ulcer and Gastro-jejunal Ulcer with a Discussion of the Indications and Contra-indications for Vagotomy. S. ARTHUR LOCALIO, PHILIP A. ZOLLER. (Erdmann Auditorium—Capacity 125).

10:30–12:30. *Traumatic Surgery.* Nonoperative Clinic: Fractures. H. H. RITTER. (Amph. D—Capacity 30).

11:30–1:00. *Oral Surgery.* Operative Clinic. F. S. DUNN. (O.R. 5—Capacity 10).

2:00–3:30. *Orthopedic Surgery.* Nonoperative Clinic: Demonstration of Orthopedic Conditions—Pre and Post Operative. GEORGE ANOPOL. (Amph. C).

2:00–5:00. *Neurosurgery.* Operative Clinic. J. A. MACLEAN. (O.R. 5—Capacity 10).

2:00–5:00. *Traumatic Surgery.* Operative Clinic. H. H. RITTER. (Surg. Amph.—Capacity 50).

PRESBYTERIAN HOSPITAL

8:00–12:00. *General Surgery.* Operative Clinic: Surgery of the Pancreas. JOHN S. LOCKWOOD, WILLIAM B. PARSONS. (19th Fl., Main O.R. C–H—Capacity each 16).

Surgery of the Spleen. LOUIS M. ROUSSELOT, ROBERT H. E. ELLIOTT. (Same Rm.).

Resection of Maxilla For Carcinoma. MAURICE J. HICKEY. (Same Rm.).

Lobectomy for Chronic Bronchiectasis. RICHMOND L. MOORE. (Same Rm.).

2:00–4:00. *General Surgery.* Nonoperative Clinic: Tissue Culture as an Adjunct to Surgery. MARGARET MURRAY. (McCosh Amph. 18th Fl.—Capacity 142).

Phlebograms of the Lower Extremity. DAVID C. BULL. (Same Rm.).

Oxidized Cellulose in Surgery. VIRGINIA K. FRANTZ. (Same Rm.)

Electrophoretic Studies of Plasma in Extensive Burns. JOHN SCUDDER. (Same Rm.).

Indications for Portacaval Shunt. ARTHUR H. BLAKEMORE. (Same Rm.).

PRESBYTERIAN HOSPITAL BABIES HOSPITAL

9:00–12:00. *General Surgery.* Operative Clinic: Operative Clinic and Ward Rounds. Members of Staff. (O.R. 1 & 2, 9th Fl.—Capacity each 5).

2:00–4:00. *General Surgery.* Nonoperative Clinic:

Congenital Diaphragmatic Hernia. Follow-up. EDWARD J. DONOVAN. (Amph. 4th Fl.—Capacity 75).

An Analysis of the Neoplasms in Childhood Encountered at Babies Hospital in the Past Ten Years. DOROTHY H. Andersen.

Meconium Ileus. Presentation of Cases. Motion Pictures Illustrating Pathology and Operative Procedure. ROBERT B. HIATT.

Acute Appendicitis in Children. A Comparison of the Last Two Five-Year Periods with and without Chemotherapy. EARL C. TAYLOR.

PRESBYTERIAN HOSPITAL SLOANE HOSPITAL FOR WOMEN

9:00–10:30. *Obstetrics and Gynecology.* Operative Clinic: Total Hysterectomy. E. COLER. (O.R. 17th Fl.—Capacity 20).

10:30–12:30. *Obstetrics and Gynecology.* Operative Clinic: Myomectomy. JAMES CORSCADEN. (Same Rm.).

2:00–3:00. *Obstetrics and Gynecology.* Nonoperative Clinic: Rh Factor in Obstetrics. J. GREER. (Lecture Rm., 17th Fl.—Capacity 40).

3:00–4:00. *Obstetrics and Gynecology.* Nonoperative Clinic: Review of Cardiac Deaths During Pregnancy: 1925–47. A. J. TILLMAN. (Same Rm.).

PRESBYTERIAN-NEW YORK ORTHOPEDIC HOSPITALS

Fracture Service

9:00–12:00. *Fractures and Traumatic Surgery.* Operative Clinic. FRACTURE SERVICE STAFF. (Main O.R., 19th Fl.—Capacity 8).

1:30–4:00. *Fractures and Traumatic Surgery.* Nonoperative Clinic: Complicated Elbow Fractures with Follow-up Results. FREDERICK M. SMITH. (Rm. 417, 16th Fl. Phys. & Surg. Bldg.—Capacity 100).

Replacement of Articular Elements. STEPHEN S. HUDACK. (Same Rm.).

ROOSEVELT HOSPITAL

2:00–5:00. *Genitourinary.* Operative Clinic: Suprapubic Prostatectomy. SIMON A. BEISLER and STAFF. (O.R. 1–4—Capacity 56).

Nonoperative Clinic: The Presentation of Patients, Illustrating the Results of Urological Procedures. Total Cystectomy With Skin and Sigmoidal Implantation. SIMON A. BEISLER and STAFF. (Conf. Rm. 1—Capacity 50).

The Reimplantation of Skin Ureterostomies Into the bowel. SIMON A. BEISLER and STAFF. (Same Rm.).

ST. LUKE'S HOSPITAL

8:30–11:00. *General Surgery.* Operative Clinic: General Surgery. WILLIAM MACFEE and STAFF. (Lyle O.R.—Capacity 30).

11:00–1:00. *General Surgery.* Clinical Demonstrations: Benign Intrathoracic Tumors. ALEXANDER ADA. (Staff Conference Rm.—Capacity 60).

Cervical Discs. HERBERT PARSONS. (Same Rm.).

Carcinoma of the Lower Sigmoid and Upper Rectum. WILLIAM MACFEE. (Same Rm.).

Trends in Curare—Technique Anesthesia. EDWARD BURFORD and STAFF. (Same Rm.).

Carcinoma of Colon—Ten-year Study. EDWARD DONOVAN. (Same Rm.).

Management of Hemorrhage for Gastric and Duodenal Ulcers. ROBERT FRASER. (Same Rm.).

2:00–4:00. *Ophthalmology.* Operative Clinic. GUERNSEY FREY and STAFF. (Lyle O.R.—Capacity 6).

4:00–5:00. *Ophthalmology.* Clinical Demonstrations: Glaucoma. Results of Operation. GUERNSEY FREY. (Staff Conference Rm.—Capacity 60).

Extraocular Muscle Surgery. Follow-up Results. HENRY SMITH. (Same Rm.).

ST. VINCENT'S HOSPITAL

9:00–12:00. *Gynecology and Obstetrics.* Operative Clinic: Vaginal Plastics. Abdominal Hysterectomy. Vaginal Hysterectomy. Cesarean Section. JOHN MCGRATH, JAMES P. HENNESSY, THOMAS E. LAVELL, JOSEPH E. CORR, THOMAS IOVINO, ROBERT LOWRIE, JOHN

KILGUS, WALTER P. GAGE. (O.R. C & D—Capacity each 10).

10:00–12:00. *General Surgery.* Nonoperative Clinic: Round Table Conference: Use of Transverse Incisions. JOHN A. LAWLER. (Nurse's Lecture Rm.—Capacity 60).
Malignant Tumors Small Intestine. CLARENCE P. HOWLEY. (Same Rm.).

10:00–12:00. *Radiology.* Nonoperative Clinic: Roentgen Diagnosis of Lesions of Small Intestines. Differential Diagnosis of Mechanical and Adynamic Ileus. WALLACE MAYER and ASSOCIATES. (Viewing Rm.—Capacity 25).

2:00–3:00. *Gynecology and Obstetrics.* Nonoperative Clinic: Round Table Conference:
Study of Vaginal Spread for Carcinoma of Cervix. Differential Diagnosis in Cases of Acute Pelvic Pathology. Indications for Cesarean Section. Choice of Operation in Uterine Prolapse. JOHN MCGRATH and ASSOCIATES. (Nurse's Auditorium—Capacity 150).

3:00–4:00. *Pathology.* Nonoperative Clinic: Study of Abdominal and Chest Fluids for Evidence of Malignancy. Display of First Pathological Material. Display and Demonstration of System Diseases by Kodachrome Slides. A. ROTTINO and ASSOCIATES. (Same Rm.).

4:00–5:00. *General Surgery.* Nonoperative Clinic: Thrombosis and Embolism. LOUIS F. SANMAN. (Same Rm.).

SLOANE HOSPITAL FOR WOMEN
(See Presbyterian Hospital)

U. S. MARINE HOSPITAL
(STATEN ISLAND)

9:00–10:00. *Orthopedic Surgery.* Nonoperative Clinic: Orthopedic Conference. A. A. MICHELE and STAFF. (Doctor's Staff Rm.—Capacity 75).

10:00–12:30. *General Surgery.* Nonoperative Clinics: H. L. SKINNER, CHARLES FERGUSON and STAFF.
Regional Ileitis—Discussion and Case Presentation. (Doctor's Staff Rm.—Capacity 75).
Benign Lesion of Colon—Discussion and Case Presentation. (Same Rm.).
Malignant Lesion of Colon—Discussion and Case Presentation. (Same Rm.).

12:30–1:00. Luncheon at the Hospital.

1:00–3:00. *General Surgery.* Operative Clinic: Stomach Resection Under Balanced Anesthesia. W. F. NICKEL and RESIDENT STAFF. (General O.R.—Capacity 10).

1:00–3:00. *Orthopedic Surgery.* Operative Clinic: Herniated Intervertebral Disc with Exploration and Fusion. H. L. SKINNER, A. A. MICHELE and STAFF. (General O.R.—Capacity 10).

VETERANS ADMINISTRATION HOSPITAL

9:00–12:00. *General Surgery.* Operative Clinic. EDWIN SELF, DAVID K. PINKS. (Bldg. D, 2 Rms., 12th Fl.—Capacity 20).

9:00–12:00. *Neurosurgery.* Operative Clinic: Prefrontal Lobotomy. JOHN E. SCARFF, ERICH KRUEGER. (Bldg. D, 12th Fl. 1 Rm.—Capacity 10).

9:00–12:00. *Genitourinary Surgery.* Operative Clinic. LOUIS SPIVACK, EDWIN J. DREW. (Bldg. D, 12th Fl., 1 Rm.—Capacity 10).

9:00–12:00. *Plastic Surgery.* Operative Clinic. HERBERT CONWAY, CORNELIUS J. KRAISSL. (Bldg. B., 5th Fl., 1 Rm.—Capacity 10).

9:00–12:00. *Orthopedic Surgery.* Nonoperative Clinic: Conference: Presentation of End-Results—Bone Tumors; Bone and Joint Tuberculosis. Presentation of Cases. PHILIP D. WILSON, SYDNEY N. EICHENHOLTZ, ROBERT PATTERSON, FRANK E. STINCHFIELD, and RESIDENT STAFF. (Bldg. C, 4th Fl.—Capacity 30).

9:30–11:30. *General Surgery.* Nonoperative Clinic: Medical Rehabilitation Clinic. HARRY KESSLER, KARL HARPUDER, IRWIN D. STEIN, BERNARD STOLL, JACOB BYER. (Bldgs. B, C, & F, Ground Fl.—Capacity 20).

11:00–12:30. *General Surgery.* Nonoperative Clinic: Result of Vagus Resection. P. K. SAUER, JOHN ECKEL, CRANSTON HOLMAN, FRANK J. MCGOWAN, CHARLES PRIVITERI, and RESIDENT STAFF.

11:00–12:30. *Neurosurgery.* Nonoperative Clinic: Prefrontal Lobotomy. JOHN E. SCARFF, THOMAS RENNIE, HILAND L. FLOWERS.

1:30–3:30. *Neurosurgery.* Nonoperative Clinic: Conference: Complications in Frontal Cranioplasties Involving Frontal Sinus. Surgery of the Sympathetic Nervous System for Painful Conditions. Leptomeningeal and Porencephalic Cyst with Increased Intracranial Pressure. Surgical Treatment of Phantom Limb. Compression of the Anterior Pyramidal Tract by Cervical Midline Disc. Luetic Pachymeningitis with Increased Intracranial Pressure. JOHN E. SCARFF, FRITZ CRAMER, E. G. KRUEGER, J. LAWRENCE POOL. (Bldg. A, 3rd Fl.—Capacity 200).

2:00–4:00. *Otorhinolaryngology.* Operative Clinic. PETER D. LATELLA. (Bldg. B, 5th Fl.—Capacity 5).

3:30–5:00. *Urology.* Nonoperative Clinic: Conference. ARCHIE DEAN, LOUIS SPIVACK, PERRIN SNYDER, EDWIN J. DREW, WILLET WHITMORE, JOHN K. LATTIMER, and RESIDENT STAFF. (Bldg. A, 3rd Fl.—Capacity 200).

EXHIBITS
(Bldg. A, 3rd Fl.)

Anesthesia Section
Bronchogenic Carcinoma: Review of 600 Cases
Intracranial Neoplasms
Residency Training Program
Organization of Surgical Division
Results of Vagus Resection
Pathology Section
Medical Illustrations Laboratory
Plastic Surgery
Prosthetic Appliances

CLINICS IN BROOKLYN—LONG ISLAND HOSPITALS

Thursday

BROOKLYN EYE AND EAR HOSPITAL

9:00–12:00. *Ophthalmology, Otolaryngology and Faciomaxillary Surgery.* Late Results in Faciomaxillary Surgery. Late Results in Laryngectomies. Late Results in Glaucoma Surgery. Angioscotometry Exhibit. Motion Picture Demonstration on the Development of the Human Eye as Shown by Animation. G. R. O'BRIEN, JOHN P. BAKER, CHARLES LABRIOLA, JOHN EVANS, G. H. PAFF (Ph.D.). (Solarium—Capacity 40).

2:00–4:00. *Plastic Surgery.* Operative Clinic: Nasal. GERALD R. O'BRIEN and STAFF. (7th Fl. O.R. 1–4, Capacity 20).

CUMBERLAND HOSPITAL

9:00–12:00. *Fractures and Traumatic Surgery.* Operative and/or Nonoperative Clinics: Pinning for Femur Neck Fracture. LEO FASKE and STAFF. (Auditorium—Capacity 300).

Late Results in Fracture of Femur Neck. (Same Rm.).
Traumatic Abdomen. JOSEPH I. ANTON. (Same Rm.).
Ward Rounds with X-ray Studies of Various Types of Fractures. LEO FASKE and STAFF. (Wards 46, 51—Capacity 15).

12:30–1:30. Luncheon at the Hospital.

2:00–5:00. *Fractures and Traumatic Surgery.* Nonoperative Clinics: Nonunion, Late Results and General Discussion. JOSEPH NEVINS and STAFF. (Auditorium—Capacity 300).
Ward Rounds with Demonstration of Apparatus in Use. JOSEPH NEVINS and STAFF. (Wards 46, 51—Capacity 15).
Gadgets and Their Use in Fracture Treatment. JOSEPH NEVINS and STAFF. (Same Rm.).
Head Injuries. Late Results. FRANK TURNEY. (Auditorium—Capacity 300).

HOUSE OF ST. GILES THE CRIPPLE

11:00–1:00. *Orthopedic Surgery.* Operative Clinic: Abduction Traction Splint for Congenital Dislocation of Hip. C. C. VITALE (Roof Clinic—Capacity 60).
Shoulder Fusion, for Polio Flail Shoulders; five-year Follow-up; Demonstration of Cases. C. C. VITALE. (Same Rm.).
Follow-up on Shelf Operation, for Congenital Dislocation of Hip; Presentation of Cases. J. GIANQUINTE. (Same Rm.).
Recurrent Clubfoot; Demonstration of Cases Following Resection of Head of Talus Plus Transplantation of Anterior Tibial Tendon. A. OAKES. (Same Rm.).
Demonstration of Cases Following Teres Major Transplantation for Obstetrical Palsy. C. C. VITALE. (Same Rm.).

JEWISH HOSPITAL

8:00–10:00. *General Surgery.* Operative Clinic. PAUL W. ASCHNER and STAFF. (Amph. 1—Capacity 25).
Operative Clinic. LEO S. SCHWARTZ and STAFF. (Amph. 2—Capacity 25).

10:00–12:00. *Gynecology and Obstetrics.* Nonoperative Clinic:
Puerperal Hematomas of Vulva. PAUL PEDOWITZ. (Auditorium—Capacity 200).
Subcutaneous Heparin in Thromboembolic Disease in Gynecology and Obstetrics. HARRY J. GREENE. (Same Rm.).
A Study of Pelvic Drive in Obstetrics. EDWIN M. GOLD. (Same Rm.).
Hyaluranidase and Infertility. CHARLES BIRNBERG.
"Failed Forceps." ISADORE DAICHMAN, WILLIAM POMERANCE. (Same Rm.).
Studies with Gonadogen. SAMUEL A. WOLFE, IRWIN NEIGUS. (Same Rm.).

10:00–12:00. *Gynecology and Obstetrics.* Nonoperative Clinic:
Diagnosis and Treatment of Solitary Cyst of the Kidney by Aspiration and Injection. ABRAHAM D. SEGAL. (Conference Rm.—Capacity 40).
Treatment of Hydrocele by Sclerosing Agents. LOUIS H. BARETZ. (Same Rm.).
Periostitis and Ostitis Pubis Complicating Urologic Procedures. ZACHARY R. COTTLER, SAMUEL PARNASS. (Same Rm.).
Obstructive Uropathy in Children. PERRY KATZEN. (Same Rm.).
Serum Acid Phosphatase in the Diagnosis and Management of Carcinoma of the Prostate. ISADORE KIMMEL, PAUL W. ASCHNER. (Same Rm.).

KINGS COUNTY HOSPITAL

8:30–1:00. *Neurosurgery.* Operative Clinics:
Dorsal Cordotomy for Painful Phantom Foot. E. JEFFERSON BROWDER. (B. Bldg. O.R. 1–10—Capacity 15).
Sympathectomy for Arterial Hypertension. EVERETT CORRADINI. (Same Rm.).
Capsular Fiber Section for Parkinsonism. E. JEFFERSON BROWDER. (Same Rm.).
Dorsal Rhizotomy for Spasticity of Cerebral Palsy. FRANK TURNEY. (Same Rm.).

9:00–1:00. *General Surgery.* Operative Clinic. JOSEPH TENOPYR and STAFF. (B Bldg. O.R. 1–10—Capacity 15).

9:00–1:00. *Thoracic Surgery.* Operative Clinic: Segmental Resection of the Lung. LEW A. HOCHBERG. (B Bldg. O.R.F.—Capacity 15).

2:00–5:00. *General Surgery.* Nonoperative Clinic: Follow-up Fractures. JOSEPH TENOPYR. (Main Bldg. Rm. B3—Capacity 50).

2:00–5:00. *Neurosurgery.* Nonoperative Clinics:
The Cerebellar Hemangioblastomas. EVERETT CORRADINI. (Main Bldg. Lecture Rm. 1st Fl.—Capacity 40).
Cysticercus Disease of the Brain. LESTER REED. (Same Rm.).
Surgery for Parkinson's Disease. E. JEFFERSON BROWDER. (Same Rm.).
Management of Fractures of the Spine with Spinal Cord Damage. FRANK TURNEY. (Same Rm.).
Subdural Hematoma. RICHARD GRIMES. (Same Rm.).

2:00–5:00. *Thoracic Surgery.* Nonoperative Clinics:
Lung Abscess as Seen at the Kings County Hospital. ALFRED D. BICUNAS. (F Bldg. Chapel—Capacity 100).
Pneumonolysis in Cases of Pulmonary Tuberculosis. EDWIN H. GRIFFIN. (Same Rm.).
Some Experiences in the Management of Chronic Empyema. ELGIE K. JOHNSON. (Same Rm.).
Paradoxical Respiration—Its Causes, Consequences and Treatment. LEW A. HOCHBERG. (Same Rm.).

KINGS COUNTY HOSPITAL
LONG ISLAND COLLEGE DIVISION

8:30–1:00. *General Surgery.* Operative Clinics:
Gastric Resection. EDWARD P. DUNN. (B Bldg. O.R. 1–10—Capacity 20).
Large Bowel Resection. CHARLES B. JONES. (Same Rm.).
Gall Bladder and Common Duct Exploration. PHILIP E. LEAR. (Same Rm.).
Ligation Patent Ductus Arteriosus. JOHN L. MADDEN. (Same Rm.).

1:45–5:00. *General Surgery.* Nonoperative Clinics:
Diagnosis and Treatment of Lymphogranuloma Venereum, Anus, Rectum, and Colon. CHARLES B. JONES. (Nurse's Home Classroom—Capacity 50).
Paraduodenal Hernia, Report of Four Cases. PHILIP E. LEAR. (Same Rm.).
Cosmetic Incision for Excision of Benign Breast Tumors and for Suppurative Diseases of the Female Breast. GAETANO T. DEYOANNA. (Same Rm.).
Tetanus—A Surgical Problem. Experience with Five Hundred Cases in China. PHILLIPS F. GREENE. (Same Rm.).
Gastrojejunocolic Fistula. Six Case Reports. JOHN L. MADDEN. (Same Rm.).
Follow-up Results in Hip Nailing Operations in Femoral Neck. ARNOLD F. SAMMIS. (Same Rm.).

LONG ISLAND COLLEGE HOSPITAL

9:00–12:30. *Obstetrics and Gynecology.* Operative and Nonoperative Clinics, and Demonstrations. Members of the staff. (Surg. Amphitheater—Capacity 100).
The Departments of Ophthalmology and Otolaryngology will conduct their programs at the Brooklyn Eye and Ear Hospital.

METHODIST HOSPITAL

9:00–12:00. *Obstetrics and Gynecology.* Operative Clinics: Vaginal Hysterectomy. Total Hysterectomy. (Other Operative Procedures to be Scheduled Later). G. H. DAVIS and STAFF. (Buckley Bldg.—8th Fl., Main O.R. 1–6—Capacity 50).
9:00–12:00. *Obstetrics and Gynecology.* Demonstrations:
Ball Technique in X-Ray Mensuration of Pelvis. J. I. BUTLER, E. GOYANES. (Maternity Bldg.—Main Fl., Prenatal Clinic—Capacity 40).
Cord Transfusion. B. MAYES. (Same Rm.).
Inspection of Clinics, Wards and Delivery Rooms. RESIDENT STAFF. (Same Rm.).
Interesting Cases. B. A. G. WEISL.
Maternal Mortality and Morbidity in Obstetrics. I. SEISMAN. (Same Rm.).
12:00–1:00. Luncheon at the Hospital.
2:00–5:00. *Obstetrics and Gynecology.* Nonoperative Clinic: G. H. DAVIS, Chairman.
Fibroids and Ovarian Tumors Complicating Pregnancy. G. SHEPLER. (Nurse's Auditorium—Capacity 300).
Fetal Mortality in Prolonged Labor. M. C. GLYNN. (Same Rm.).
Leimyosarcoma; Contract with Cellular Myofibromyomas. W. G. FRENCH, J. S. HOWE. (Same Rm.).
Ablatio Placentae. A. R. BEIL. (Same Rm.).
Manual Removal of Placenta. H. MAYES. (Same Rm.).
Anesthesia in Cesarean Section. H. ACKEN, JR. (Same Rm.).
Wedge Resection of Ovary for Polycystic Disease. S. C. HALL. (Same Rm.).

CLINICS IN NEW YORK HOSPITALS

Friday

BEEKMAN-DOWNTOWN HOSPITAL

9:30–10:45. *Traumatic Surgery.* Nonoperative Clinic: Ward Fracture Rounds. ROBERT H. KENNEDY and STAFF. (Wards in groups of 5–6. Total Capacity—30).
10:45–12:00. *Traumatic Surgery.* Nonoperative Clinic: Case Presentation of Late Bad Results in Fractures. ROBERT H. KENNEDY and STAFF. (3rd Fl. Annex—Capacity 30).

BELLEVUE HOSPITAL—FIRST (COLUMBIA UNIVERSITY) SURGICAL DIVISION

8:00–12:00. *Thoracic Surgery.* Operative Clinic. FRANK B. BERRY and STAFF. (C & D Bldg., 5th Fl.—Capacity 10).

BELLEVUE HOSPITAL—THIRD (NEW YORK UNIVERSITY) SURGICAL DIVISION

8:00–12:00. *Anesthesia.* Demonstration: Anesthesia in Thoracic Surgery. E. A. ROVENSTINE and STAFF. (C & D Bldg., 5th Fl.—Capacity 5).

BELLEVUE HOSPITAL—FOURTH (NEW YORK UNIVERSITY) SURGICAL DIVISION

8:00–9:30. *General Surgery and Fractures.* Operative Clinics. (O.R. K-5 & K-6): ARTHUR MCQUILLAN and STAFF. (Capacity, each operating room, 5).
Thyroid Surgery. ARTHUR S. MCQUILLAN (O.R. A).
Stomach Surgery. J. WILLIAM HINTON. (O.R. B).
Intestinal Surgery. LESTER BREIDENBACH. (O.R. C).
Gall-Bladder Surgery. EDWARD V. DENNEEN (O.R. D).
Smith-Petersen Nailing for Fracture of Neck of Femur. KENNETH M. LEWIS (Bone Room K-6).
10:00–12:00. *General Surgery and Fractures.* Nonoperative Clinics: End-Results. ARTHUR S. MCQUILLAN and STAFF. (I & K Bldg., Stewart Amphitheater—Capacity 100).
Thyroids. ARTHUR S. MCQUILLAN.
Peptic Ulcers. J. WILLIAM HINTON.
Fracture of the Neck of the Femur. KENNETH M. LEWIS.
Stricture of Rectum Due to Lymphogranuloma Venereum. LESTER BREIDENBACH.
Gall-Bladder Disease. EDWARD V. DENNEEN.

BETH ISRAEL HOSPITAL

9:00–12:00. *Orthopedic Surgery.* Nonoperative Clinic. IRVIN BALENSWEIG and STAFF. (3rd Fl. Lecture Rm.—Capacity 30).
2:00–5:00. *Obstetrics.* Nonoperative Clinic. EDWIN G. LANGROCK, SAMUEL J. SCADRON. (Same Rm.).
2:00–5:00. Nonoperative Clinic: Tumor Clinic. ALFRED PLAUT and STAFF. (Auditorium—Capacity 200).

FLOWER AND FIFTH AVENUE HOSPITALS

9:00–10:00. *General Surgery.* Nonoperative Clinic: Water Balance, etc. FRANCIS D. SPEER, JOHN HERRLIN. (Auditorium—Capacity 300).
Lyophilized Amino Acids. MICHAEL G. MULINOS. (Auditorium—Capacity 300).
Relationship of Amino Acid Composition of Foods. R. J. BLOCK, H. H. MITCHELL. (Auditorium—Capacity 300).
11:00–12:00. *General Surgery.* Nonoperative Clinic: The Prevention of Renal Complications by Therapy with Mixtures of Sulfonamides—or—Phthalys Sulfacetamide in Surgery of Colon. DAVID LEHR. (Auditorium—Capacity 300).
2:00–3:00. *General Surgery.* Nonoperative Clinic: Presentation of Plans for Resident Training in Surgery. JAMES M. WINFIELD. (Auditorium—Capacity 300).

HARLEM HOSPITAL

9:00–12:00. *General Surgery.* Operative and Nonoperative Clinic: Abdominal Catastrophies. B. N. BERG, A. A. ZINGARO, J. L. WILSON, A. DEL. MAYNARD. (Main Bldg. O.R.—Capacity 25 or/and Gyn. O.R. Women's Pavilion—Capacity 10 & Conference Rm. 5th Fl. Women's Pavilion—Capacity 25). (Ward Rounds will be held on 2D and 5W).
2:00–5:00. *General Surgery.* Nonoperative Clinic: Clinicopathological Conference. L. T. WRIGHT, S. WEINTRAUB and STAFF. (Conference Rm. 5th Fl. Women's Pavilion—Capacity 100).

HOSPITAL FOR JOINT DISEASES

9:00–10:30. *General Surgery.* Operative Clinic: Carcinoma of Colon. Carcinoma of Rectum. Nonoperative Clinic:

Intra-Abdominal Resection with Anastomosis for Carcinoma of the Rectum. A. J. BELLER. (Surg. Amphitheater—Capacity 50).

2:00–4:00. *Neurosurgery.* Operative Clinic: Operations of Brain and Spinal Cord. ABRAHAM KAPLAN. (Same Rm.).

HOSPITAL FOR SPECIAL SURGERY

8:30–12:00. *Orthopedic Surgery.* Operative Clinic. Adult Service. PHILIP D. WILSON, R. STEPHENS and STAFF. (Main O.R.—Capacity 32).

2:00–5:00. *Orthopedic Surgery.* Nonoperative Clinic:

Osteoid Osteoma. MILTON HELPERN, RAYMOND LEWIS. (Lecture Hall—Capacity 100).

Bone Grafts without Internal Fixation. PETER RIZZO.

Bridging of Defects of Long Bones of the Extremities. JOHN FLANAGAN.

The Bone Bank. PHILIP D. WILSON.

Spine Fusion with Internal Fixation. L. RAMSAY STRAUB.

Problems in the Treatment of Scoliosis. JOHN R. COBB.

LENOX HILL HOSPITAL

9:00–11:00. *Gynecology.* Operative Clinic. ROYAL VAN ETTEN and STAFF. (O.R. 1, 2 & 3—Capacity each 15).

11:00–12:00. *Gynecology.* Nonoperative Clinic: Hysterectomy, Combined Method with Lantern Slides. MORTIMER RODGERS. (Einhorn Auditorium—Capacity 450).

2:00–5:00. *Orthopedic Surgery.* Nonoperative Clinics: Late Results of Unusual Cases:

Osteoarthritis of the Hip in the Aged Treated by Bifurcation. Surgical Treatment of Corns. WALTER GALLAND. (Einhorn Auditorium—Capacity 450).

Spinal Fusion for the Unstable Spine Giant Cell Tumors of Bone. WALTER SWIFT. (Same Rm.).

Combined Internal-External Splinting of Fractures by the Roger-Anderson Technique. HENRY JORDAN. (Same Rm.).

Bilateral Epiphseolysis Capitus Femoris (Slipping of the Upper Femoral Epiphyses. FRANCIS ROTH. (Same Rm.).

Inspection of Lenox Hill Brace Shop.

LYING-IN HOSPITAL

(See New York Hospital)

MANHATTAN EYE, EAR AND THROAT HOSPITAL

9:30–12:00. *Otolaryngology.* Nonoperative Clinic: Demonstration: Audiometry; Selection and Fitting of Hearing Aids; Retraining Residual Hearing. WHITEHURST, NERENBERG. (Jones Clinic, 2nd Fl.—Capacity 24).

10:00–12:00. *Ophthalmology.* Nonoperative Clinic: Demonstration: Corneal Grafting Technique on Rabbits. H. M. KATZIN. (Eye Bank Lab., 10th Fl.—Capacity 12).

10:00–12:00. *Neurosurgery.* Operative Clinic. Facial Nerve Graft:

Operation and Demonstration of Postoperative Results. T. G. TICKLE. (O.R. 6th Fl.—Capacity 5).

2:00–4:00. *Otolaryngology.* Operative Clinic. Tonsil Surgery; Preoperative and Postoperative Procedure. R. J. BELLUCCI. (O.R. 7th Fl.—Capacity 5).

2:00–5:00. *Ophthalmology.* Nonoperative Clinic: Orthoptic Clinic. F. C. KEIL, JR., E. KNAUBER. (1st Fl. Rear—Capacity 12).

MEMORIAL HOSPITAL

9:00–10:30. *Tumor Surgery.* Nonoperative Clinic: End-Results Following Abdominoperineal Resection for Cancer of the Rectum; Treatment of Cancer of the Colon; Management of Squamous Cancer of the Anus and the Rectum. G. E. BINKLEY. (Auditorium—Capacity 250).

10:30–12:00. *Tumor Surgery.* Nonoperative Clinic: End-Results in the Treatment of Cancer of the Stomach; a Study of Over 1000 Cases. End-Results in the Treatment of Malignant Melanomas; a Study of Over 900 Cases. GEORGE T. PACK, ISABEL M. SHARNAGEL. (Same Rm.).

2:00–3:00. *Thoracic Surgery.* Nonoperative Clinic: Asymptomatic Thoracic Tumors: End-Results in Surgically Treated Cases of Carcinoma of the Cervical Esophagus. End-Results in Surgically Treated Cases. W. L. WATSON. (Same Rm.).

3:00–4:00. *Tumor Surgery.* Nonoperative Clinic; End-Results in Treatment of Bone Tumors. B. L. COLEY. (Same Rm.).

4:00–5:00. *Tumor Surgery.* Nonoperative Clinic: Discussion: Cancer in Children; Presentation of Followed Cases. H. W. DARGEON. (Same Rm.).

METROPOLITAN HOSPITAL

9:00–12:00. *General Surgery.* Operative Clinic: Surgery of Stomach and Duodenum. JAMES M. WINFIELD and STAFF. (Conference Rm.—Capacity 60).

Indications. EDWARD J. MCCABE. (Same Rm.).

Vagotomy. JAMES M. WINFIELD. (Same Rm.).

Duodenal Closure and Complications. KENNETH C. PEACOCK. (Same Rm.).

Surgery of Gall Bladder and Pancreas. KENNETH C. PEACOCK. (Same Rm.).

Retroperitoneal Tumors. KENNETH C. PEACOCK, JOHN SNIDER. Same Rm.).

Peritoneal Spaces. JOHN HERRLIN. (Same Rm.).

2:00–4:00. *Plastic and Faciomaxillary Surgery.* Operative and Nonoperative Clinic: Plastic Clinic. DAVID M. MAYER, JOHN F. FORD. (Main O.R. 1—Capacity 30).

MOUNT SINAI HOSPITAL

8:30–12:00. *Genitourinary Surgery.* Operative Clinic: Kidney Operation. LEO EDELMAN. (Surg. Amphitheater—Capacity 100).

Nonoperative Clinic: Carcinoma in a Horseshoe Kidney. GORDON D. OPPENHEIMER. (Same Rm.).

Bladder Neck Obstruction in Young Individuals. MOSES SWICK. (Same Rm.).

Perirenal Insufflation. WILLIAM MENCHER. (Same Rm.).

Enuresis Due to Ectopic Ureter. H. EVANS LEITER. (Same Rm.).

NEUROLOGICAL INSTITUTE

(See Presbyterian Hospital)

NEW YORK EYE AND EAR INFIRMARY

10:00–12:00. *Otolaryngology.* Demonstration: Anatomical Specimens. J. SWIFT HANLEY. (Weeks Hall—Capacity 100).

2:00–4:00. *Otolaryngology.* Discussion of Pathology with Lantern Slides. D. S. D. JESSUP. (Same Rm.).

The routine clinics of the New York Eye & Ear Infirmary will be held as usual during this period; all Fellows of the American College of Surgeons are cordially invited to attend.

NEW YORK HOSPITAL

9:00–12:00. *Orthopedic Surgery.* Operative Clinic. FREDERICK LEE LIEBOLT. (10th Fl. O.R., 1 Rm.—Capacity 30).

2:00–5:00. *Orthopedic Surgery.* Nonoperative Clinic. FREDERICK LEE LIEBOLT and ASSOCIATES.

Segmental Arterial Spasm Associated with Supracondylar Fracture of the Elbow (Case Report). ARTHUR CONSOLE. (Rm. F-639—Capacity 70).
Reconstruction Surgery of the Hand (Lantern Slides). FREDERICK L. LIEBOLT. (Same Rm.).
Massive Onlay Bone Grafts for Ununited Fractures. PRESTON WADE. (Same Rm.).
Conservative Treatment of Table Fractures of the Tibia. BERNARD MAISEL. (Same Rm.).
End-Results in Treatment of Fractures of the Neck of the Femur by Moore Nails. NELSON CORNELL. (Same Rm.).

2:00–5:00. *Otorhinolaryngology.* Operative Clinic: Surgery of the Oropharynx and Nasopharynx. THOMAS GARRICK. (10th Fl. O.R., 1 Rm.—Capacity 30).

2:00–5:00. *General Surgery.* FRANK GLENN and STAFF.
The Chemistry of Penicillin and Streptomycin. VINCENT DU VIGNEAUD. (College Auditorium, Rm. B-001—Capacity 350).
Toxicity of Streptomycin. WALSH MCDERMOTT. (Same Rm.).
Treatment of Pulmonary Tuberculosis by Streptomycin. CARL MUSCHENHEIM. (Same Rm.).
Use of Streptomycin in Treating Surgical Pulmonary Tuberculosis. CRANSTON HOLMAN. (Same Rm.).
Adult Tissue Extract in Wound Healing. JAMES DINGWALL. (Same Rm.).

NEW YORK HOSPITAL
LYING-IN HOSPITAL

9:00–12:00. *Gynecology.* Operative Clinic: Gynecological Operations by RESIDENT STAFF. (Gyn. O.R., 2 Rms.—Capacity 50).

11:00–1:00. *Obstetrics and Gynecology.* Operative Clinic: Obstetrical and Gynecological Staff Rounds. GORDON DOUGLAS and STAFF. (Wards—Capacity 25).

NEW YORK ORTHOPEDIC
DISPENSARY AND HOSPITAL

8:00–12:00. *Orthopedic Surgery.* Operative Clinic. ALAN DE FOREST SMITH and STAFF. (O.R. 1 & 2—Capacity 12).

1:30–4:00. *Orthopedic Surgery.* General Orthopedic Clinic. HALFORD HALLOCK and STAFF. (7th Fl. Assembly Rm.—Capacity 30).

NEW YORK POST-GRADUATE MEDICAL SCHOOL
AND HOSPITAL

8:30–12:00. *General Surgery.* Operative Clinic. R. B. LOBBAN. (O. R. 1—Capacity 10).
Operative Clinic. J. W. HINTON. (Surg. Amphitheater—Capacity 50).

8:30–12:00. *Plastic Surgery.* Operative Clinic. GUSTAVE AUFRICHT. (O.R. 2—Capacity 10).

9:30–12:00. *Ophthalmology.* Nonoperative Clinic: Muscle Anomalies of the Eye with Demonstration of Cases. HAROLD W. BROWN. (Eye Clinic—Capacity 15).

10:30–12:30. *General Surgery.* Nonoperative Clinic: Thyroid Disease, Including Case Presentations and Discussion of Thiouricil Therapy, Carcinoma of the Thyroid, and Anatomical Consideration of the Recurrent Laryngeal Nerve. LOUIS R. SLATTERY, RALPH R. MOOLTEN. (Erdmann Auditorium—Capacity 125).

2:00–5:00. *General Surgery.* Operative Clinic. J. R. ERDMANN. (O.R. 4—Capacity 10).
Operative Clinic. C. G. HEYD. (Surg. Amphitheater—Capacity 50).

2:00–5:00. *Tumor Surgery.* Operative Clinic. H. W. MEYER. (O.R. 1—Capacity 10).

2:00–5:00. *Gynecology.* Operative Clinic. W. T. DANN-REUTHER. (O.R. 5—Capacity 10).

PRESBYTERIAN HOSPITAL

8:00–12:00. *General Surgery.* Operative Clinics: Surgery of The Large Bowel. Surgery of the Rectum (Cancer). DAVID C. BULL, ROBERT S. GRINNELL, LOUIS M. ROUSSELOT, HENRY S. COOPER. (19th Fl., Main O.R. A–H—Capacity each 16).
Vagectomy for Marginal Ulcer. ROBERT H. WYLIE. (Same Rm.).
Wiring of Arteriosclerotic Abdominal Aneurysm. ARTHUR H. BLAKEMORE. (Same Rm.).

2:00–4:00. *General Surgery.* Nonoperative Clinic: Physiology of Peritonitis. JOHN S. LOCKWOOD. (18th Fl., McCosh Amphitheater—Capacity 142).
Prevention of Wound Infection and Peritonitis with Sulfamylon and Streptomycin. Case Demonstration. EDWARD L. HOWES.
Laboratory Experiments and Clinical Results with Bacitracin. FRANK L. MELENEY.
Observations on Acute Appendicitis Over a Thirty-Year Period at a University Hospital. RUDOLPH N. SCHULLINGER.

PRESBYTERIAN HOSPITAL
NEUROLOGICAL INSTITUTE

9:00–12:00. *Neurosurgery.* Operative Clinic: Cases Previously Shown Clinically at the Monday Morning Staff Conference. Surgical Staff. (10th Fl. N.I.-O.R. 1—Capacity 12; O.R. 2—Capacity 6).

PRESBYTERIAN HOSPITAL
SLOANE HOSPITAL FOR WOMEN

9:00–10:00. *Obstetrics and Gynecology.* Nonoperative Clinic: Implantation of Radium for Carcinoma of the Cervix. JAMES CORSCADEN, S. GUSBERG. (17th Fl. Lecture Rm.—Capacity 40).

10:00–11:00. *Obstetrics and Gynecology.* Nonoperative Clinic: Late Follow-up Results in Carcinoma of the Cervix. JAMES CORSCADEN. (Same Rm.).

11:00–12:00. *Obstetrics and Gynecology.* Nonoperative Clinic: Trends in the Treatment of Uterine Carcinoma. HOWARD C. TAYLOR, JR. (Same Rm.).

PRESBYTERIAN-NEW YORK ORTHOPEDIC
HOSPITALS

Fracture Service

9:00–1:00. *Fractures and Traumatic Surgery.* Nonoperative Clinic: Fracture Service Grand Rounds and Conference. FRACTURE SERVICE STAFF. (Presb. Hosp., Ortho. Wards, 11th Fl. W.—Capacity 30).

2:00–4:00. *Fractures and Traumatic Surgery.* Nonoperative Clinic: Rôle of Sympathetic System in the Surgery of Trauma. STEPHEN S. HUDACK. (Rm. 417, 16th Fl. Phys. & Surg. Bldg.—Capacity 100).
Use of Amino Acids in Maintaining Nitrogen Balance. SIDNEY C. WERNER. (Same Rm.).
The Treatment of Shock Coincident with Trauma. JOHN SCUDDER. (Same Rm.).

ROOSEVELT HOSPITAL

9:00–12:00. *General Surgery.* Nonoperative Clinic: Tumors of the Stomach With Colored Photographs of Specimens. HOWARD A. PATTERSON. (Conference Rm. 1—Capacity 50).
Demonstration of Stomach Tumor Specimens. WALTER BRANDES. (Conference Rm. 1—Capacity 50).
Some Late Results of Conservative Treatment in Vascu-

lar Problems. GRANT P. PENNOYER. (Conference Rm. 1—Capacity 50).

The Observed Effect of Various Drugs on Intestinal Activity. (Motion Picture Demonstration). DAVID M. WEEKS. (Conference Rm. 1—Capacity 50).

ST. VINCENT'S HOSPITAL

9:00–12:00. *General Surgery.* Operative Clinics and Demonstration: Cases of Trauma Fractures. C. J. MAGUIRE, FRANK CONWAY, LOUIS SANMAN, JOHN A. FALLON, MAURICE O'SHEA. (O.R. A & B—Capacity each 5; O.R. C—Capacity 10).

9:00–12:00. *General Surgery.* Tumor Clinic and Follow-up. GEORGE R. STUART and Tumor Committee. (Nurse's Auditorium—Capacity 150).

9:00–12:00. *Neurosurgery.* Nonoperative Clinic: The Results of Conservative Treatment of Acute Head Injuries. FRANKLIN ROBINSON. (Nurse's Lecture Rm.—Capacity 60).

2:00–4:00. *Pathology.* Nonoperative Clinic: Display of Fresh Pathological Material. Display and Demonstration of Systemic Diseases by Kodachrome Slides; Display of Fixed Specimens. ANTHONY ROTTINO and ASSOCIATES. (Same Rm.).

SLOANE HOSPITAL FOR WOMEN
(See Presbyterian Hospital)

U. S. MARINE HOSPITAL
(STATEN ISLAND)

10:00–12:30. *General Surgery.* Nonoperative Clinic: Essential Hypertension—Combined Medical, Surgical, and Anesthesiology Presentation. H. L. SKINNER, C. L. HEBERT and STAFF. (Doctor's Staff Rm.—Capacity 75).

12:30–1:00. Luncheon at the Hospital.

1:00–2:00. *General Surgery.* Operative Clinic: Dorsolumbar Sympathectomy—Intratracheal Anesthesia. H. L. SKINNER and STAFF. (General O.R.—Capacity 10).

1:00–3:00. *Genitourinary Surgery.* Nonoperative Clinic: Genitourinary Surgical Ward Rounds. CHARLES FERGUSON and STAFF. (Ward E.F. 6—Capacity 20).

VETERANS ADMINISTRATION HOSPITAL

9:00–12:00. *General Surgery.* Operative Clinic. CARNES WEEKS, JOHN E. SULLIVAN. (Bldg. D, 12th Fl., 1 Rm.—Capacity 10).

9:00–12:00. *Thoracic Surgery.* Operative Clinic. WALTER CRANDALL, AARON HIMMELSTEIN, RICHMOND MOORE. (Bldg. D, 12th Fl., 1 Rm.—Capacity 10).

9:00–12:00. *Orthopedic Surgery.* Operative Clinic. SYDNEY EICHENHOLTZ, ROBERT PATTERSON, FRANK E. STINCHFIELD. (Bldg. D, 12th Fl., 1 Rm.—Capacity 10).

9:00–12:00. *Head and Neck Surgery.* Operative Clinic. EDGAR L. FRAZELL. (Bldg. D, 12th Fl., 1 Rm.—Capacity 10).

9:00–12:00. *Plastic Surgery.* Nonoperative Clinics: Plastic Surgery. HERBERT CONWAY, CLARENCE R. STRAATSMA, CORNELIUS J. KRAISSL, ROBERT H. CLIFFORD and RESIDENT STAFF.

Cases Demonstrating the Use of Large Abdominal Double-faced Flaps. (Bldg. A, 3rd Fl.—Capacity 200).

Crossed Leg Flaps. (Same Rm.).

Surgery in the Closure of Decubiti in Paraplegics. (Same Rm.).

Plastic Surgery in Cancer of the Head and Neck. (Same Rm.).

9:30–11:30. *General Surgery.* Nonoperative Clinic: Medical Rehabilitation Clinic. HARRY KESSLER, KARL HARPUDER, IRWIN D. STEIN, BERNARD STOLL, and JACOB BYER. (Bldgs. B, C, & F, Ground Fl.—Capacity 20).

11:00–12:30. *General Surgery.* Nonoperative Clinic: Thoracolumbar Sympathectomies. CARNES WEEKS. (Bldg. A, 3rd Fl.—Capacity 200).

1:30–3:30. *Radiation Therapy.* Nonoperative Clinic: Exhibit and Presentation: Bronchogenic Carcinoma—Analysis of Diagnosis and Therapy with End-Results. A Study of 600 Cases. ARCHIE SHEINMEL, BERNARD ROSWIT, JOSEPH STEIN and RESIDENT STAFF. (Bldg. A, 3rd Fl.—Capacity 200).

3:30–5:00 *Anesthesia.* Nonoperative Clinics: Conference: Endotracheal Anesthesias. Electrocardiographic Changes during Intrathoracic Surgery. EMERY ROVENSTINE, HARDOL BISHOP, CHARLES BURSTEIN and RESIDENT STAFF.

EXHIBITS
(Bldg. A, 3rd Fl.)

Anesthesia Section
Bronchogenic Carcinoma: Review of 600 Cases
Intracranial Neplasms
Residency Training Program
Organization of Surgical Division
Results of Vagus Resection
Pathology Section
Medical Illustrations Laboratory
Plastic Surgery
Prosthetic Appliances

WOMAN'S HOSPITAL

8:30–12:30. Obstetrics and Gynecology. Nonoperative Clinics: ALBERT ALDRIDGE and STAFF. (4th Fl. Conference Rm.—Capacity 75).

Treatment of Neoplasms of the Female Genital Tract.

Management of Birth Injuries and Uterine Displacements.

Diagnosis and Treatment of Obstetric Complications.

End-Results of Treatment of These Conditions Will Be Discussed.

During the course of the morning the routine for training of Residents in Gynecology and Obstetrics will be outlined.

The pathologist will give demonstrations of obstetric and gynecologic pathological specimens.

CLINICS IN BROOKLYN—LONG ISLAND HOSPITALS

Friday

CUMBERLAND HOSPITAL

9:00–12:00. *General Surgery.* Operative Clinic: Cancer and Prostate. HOWARD T. LANGWORTHY and STAFF. (O.R. 1, 2, 3—Capacity 20).

Orchiectomy for Prostatic Metastases. Late Results. LEO DREXLER. (Auditorium—Capacity 300).

Cancer of Bladder with Transplantation of Ureter. Late Results. LEO DREXLER and STAFF.

12:00–12:30. Retroperitoneal Teratoma with Slides. JOSEPH I. ANTON.

2:00–5:00. Nonoperative Clinic. Late Results in Thrombophlebitis and Phlebothrombosis. WARD CEILLY.

Tumors in Children. HERMAN CHARACHE.

Convulsions During Anesthesia. Late Results. IRVING M. PALLIN.

KINGS COUNTY HOSPITAL

9:00–1:00. *Gynecology.* (Kings County Division) Operative Clinics. GARLICK, MCGOLDRICK and MUELLER. (B Bldg. O.R. 1–10—Capacity 15).

9:00–1:00. *Thyroid Surgery.* Operative Clinic: Thyroidectomy. ANTHONY PIRUNDINI. (Same Rm.).

Nonoperative Clinic: Use of Propyl Thiouracil. (To follow in O.R.). (Same Rm.).

9:00–1:00. *General Surgery.* Nonoperative Clinic: JOHN F. RAYCROFT and STAFF.

Cholesystectomy in the Acute Gall Bladder as Compared with Cholesystectomy in the Chronic Gall Bladder. IRWIN P. TRAIN. (O.P.D. Bldg., Lecture Rm.—Capacity 100).

Use of the Miller-Abbott Tube As an Aid in Intestinal Surgery. JOHN F. RAYCROFT. (O. P. D. Bldg., Lecture Rm.—Capacity 100).

Review of Gastric and Duodenal Surgery. HARVEY F. POTTER. (Same Rm.).

Fracture of the Neck of the Femur—Five-year Study with Follow-up. VINCENT S. MAGGIO. (Same Rm.).

Case Report. Congenital Idiopathic Dilatation of the Common Bile Duct. JOHN F. RAYCROFT. (Same Rm.).

Review of Amputations of the Lower Extremities. THOMAS W. HYNES. (Same Rm.).

Review of Acute Appendicitis. PETER FIORE. (Same Rm.).

Review of Large Bowel Surgery. BERNARD J. FICARRA. (Same Rm.).

Therapy of Peripheral Vascular Disease. C. BURLING ROESCH. (Same Rm.).

2:00–5:00. *Gynecology.* Nonoperative Clinic: Follow-up of Gynecologic Malignancies. JOSEPH MCGOLDRICK. (Main Bldg., Rm. B-3—Capacity 50).

LONG ISLAND COLLEGE HOSPITAL

9:00–12:30. *General Surgery.*

Operative Program. EMIL GOETSCH and STAFF. (Surg. Amphitheater—Capacity 100).

Nonoperative Clinic: Fractures and Other Trauma. S. POTTER BARTLEY, HAROLD LAROE. (Surg. Amphitheater—Capacity 100).

Nonoperative Clinic: Diagnostic and Therapeutic Nerve-Blocks. PAUL ANSBRO. (Surg. Amphitheater—Capacity 100).

The Departments of Ophthalmology and Otolaryngology will conduct their programs at the Brooklyn Eye and Ear Hospital.

METHODIST HOSPITAL

9:00–12:00. *Tumor Surgery.* Nonoperative Clinic: Tumor Clinic: (Late Results). PIERRE A. RENAUD, Moderator, and Hospital Staff. (Nurse's Auditorium—Capacity 300).

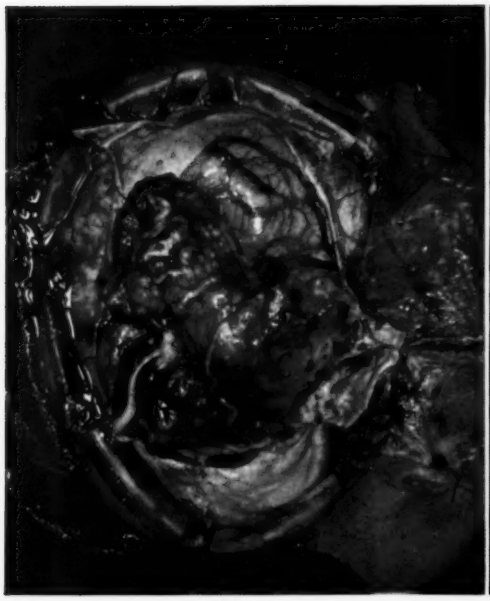

Fig. 1.

Fig. 2.

Fig. 3.

Fig. 4.

Intracranial Meningiomas, Surgical Results.—**Francis C. Grant**